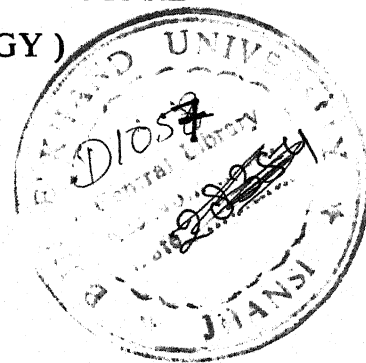


THE STUDY OF EPIDURAL ANAESTHESIA IN PAEDIATRIC SURGERY

**A
THESIS**
for

DOCTOR OF MEDICINE

(ANAESTHESIOLOGY)



**BUNDELKHAND UNIVERSITY
JHANSI (U. P.)**

1998

Sandeep Mehrotra

**Dedicated to
my parents
wife Shilpi
&
brother Ashish
&
his wife Nilima**

CERTIFICATE

This is to certify that the work entitled 'The study of epidural anaesthesia in paediatric surgery' which is being submitted as a thesis for M.D. (Anaesthesiology) has been carried out by Dr. Sandeep Mehrotra under my direct supervision and guidance.

The techniques and statistical methods used were undertaken by the candidate himself. The same were checked by me from time to time.

Date : 24.4.98


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(Supervisor)

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CERTIFICATE

This is to certify that the work entitled 'The study of epidural anaesthesia in paediatric surgery' which is being submitted as a thesis for M.D. (Anaesthesiology) has been carried out by Dr. Sandeep Mehrotra in the Department of Anaesthesiology, M.L.B. Medical College, Jhansi.

He has put in the necessary stay in the department as per university regulations.

Dated :


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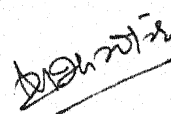
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(SANDEEP MEHROTRA)

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INTRODUCTION

INTRODUCTION

In pediatric patients, most regional blocks are performed with the primary goal of providing postoperative analgesia. A child awakening without pain is much easier to manage than one who wakes with pain; it is possible that a child who wakes up after a regional block will never develop the same level of pain that a child who wakes up in pain will experience.

Regional analgesia has been associated with earlier ambulation and discharge, as well as decreased need for both narcotic and non narcotic analgesics. Profound analgesia is produced with minimal physiologic alterations. This pain free period provides ideal psychological conditions for the recovering child and the family, and because the duration of action of most blocks is fairly predictable, administration of a subsequent analgesic drug can be precisely timed so that it becomes effective as the block wears off. Regional anaesthesia is also useful when general anaesthesia is technically difficult or is associated with an increased morbidity & mortality. Regional anaesthesia may offer an alternative to general anaesthesia in children with neuromuscular, metabolic, cardiac or chronic lung disease, with a history of malignant hyperthermia, and in emergency situations when patients are at increased risk of pulmonary aspiration of stomach contents. Regional anaesthesia provides analgesia without interfering with neurologic monitoring eg, cases of trauma in which neurological assessment remains incomplete and the vital signs are labile (particularly with accompanying head injury).

In elective surgery, the objectives differ from those in an emergency situation. Regional anaesthesia is one of several available anaesthetic techniques. It produces quick recovery from anaesthesia while maintaining a potent analgesic effect in the post operative period that can extend from 3-24 hours.

The present study has been undertaken to know the efficacy safety, advantages & disadvantages of the technique of epidural anaesthesia in paediatric age group.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

In the paediatric patients, regional analgesia must be discussed in conjunction with use of those same techniques as adjunct intraoperative anaesthesia, since most regional blocks employed in infants & children are placed after the induction of general anaesthesia for surgical procedure. This new world of pain management opened with keller's 1884 report of the local anaesthetic properties of cocaine. Regional anaesthesia was used extensively in pediatric patients, beginning with Bier's 1889 report of a spinal anaesthetic on 11 year old child and extending into the early 20th century. General anaesthesia with open drop chloroform was the technique of choice, so the introduction of spinal anaesthesia effected a considerable reduction in morbidity and mortality(Farr,1920).

By the 1950s, regional blocks were rarely employed in infants and children and infrequently performed in adults. General anaesthesia was much improved,with the addition of muscle relaxants & modern inhalational agents. Surgeons no longer administered the anesthetic as well as performing the surgical procedure.The special skills and anatomic knowledge required for facility in performing regional anesthesia for adults or children were less universal. Furthermore, many physicians believed that children suffered less pain than adults & therefore did not require postoperative analgesia(Dilworth,1987;Scott and Cousins,1988;Elander and others,1991).

By 1975 Eather noted that regional anaesthetic techniques were under utilized in paediatric patients in the united states for three major reasons: lack of experience,fear of adverse effects,and lack of patient cooperation.Then came the 1980s when the benefits of regional anaesthesia "rediscovered" in adult patients. This most recent rediscovery may be attributed to the developement of longer acting local anaesthetic agents and the "discovery" of the value of intraoperative and post-operative analgesia without narcosis.

In the 1990s, increasing expertise in regional anesthesia for adult patients, coupled with realization that infants and children do suffer pain, spurred the increasing use of paediatric regional anaesthesia (Arther and Mc Nicol, 1986, Dalens, 1989, Sethna and Berde, 1989, Rice and Hannallah 1990, Goresky 1991, AHCPR 1992, Rice and Britton, 1992) While lack of cooperation by paediatric patients will never be eliminated, improved sedation agents and the recognition that regional anaesthesia with a light general anaesthetic as sedation is both safe and efficacious has allowed more children to receive the benefit of this - approach to balanced anaesthesia (Brown and Schulte - Steinberg 1988, Rice and Britton, 1994, Sethna and Berde, 1994).

In pediatric patients, most regional blocks are performed with the primary goal of providing postoperative analgesia (Rice and Britton 1993) A child awakening without pain is much easier to manage than one who wakes with pain; it is possible that a child who wakes up after a regional block will never develop the same level of pain that a child who wakes up in pain will experience (Langer and others, 1987, Scott, 1989).

Parental acceptance of regional anaesthetic techniques in children is very high. Broadman and Hannallah (1985) reviewed 2 years experience with 687 children who received blocks as part of their anaesthetic. More than 10% were less than 1 year of age and 60% were less than 6 years old. 90% had regional anaesthesia supplemented with light general anaesthesia, whereas 10% had a block technique combined with intravenous sedation. Two hundred families were selected at random for phone interview 3-9 months after the anaesthetic, 90% of the parents would allow their child to have another regional anaesthetic.

Pediatric patients are frequently not psychologically suitable for regional anaesthesia as the sole anaesthetic technique. Dalens and Hasnouai (1989) noted in their study of 750 children undergoing caudal block that conscious children tolerated surgery poorly from a psychologic point of view, although they were free of pain.

EPIDURAL SPACE

GROSS ANATOMY

The epidural space, also called the extradural or peridural space, is situated in the spinal canal between the dura mater and the periosteum. (Bromage PR 1978) it extends from the foramen Magnum above down to the sacral hiatus below. Above the foramen magnun, the spinal dura mater & the periosteum fuse to form the endosteal & the meningeal layers of the carebral dura. Thus, the epidural space is absent here.

The epidural space is cylindrical in shape & surrounds the subarachnoid space. The boundaries of epidural space are as follows:

- Anteriorly situated is the posterior longitudinal ligament, which is firmly adherent to the posterior aspect of the vertebral bodies and the intervertebral discs.
- Laterally situated are the pedicles of the vertebrae and the intervertebral foramina through which the epidural space sends extensions around the spinal nerves and roots.
- Posteriorly situated are the periosteum covering the anterior surfaces of the vertebral laminae & the ligamentum flavum.
- Medially, the epidural space covers the dura mater upto the body of L3 in a neonate; L1 after the first year of life.

In cross section the spinal canal is triangular in shape, & the spinal cord is elliptical in shape. Since the epidural space is present between the two, its widest part is in the midline.

Typically, the epidural space is considered to be an open space, and infact, when local anaesthetic solution is injected, the distribution of analgesia is uniform in most cases.

Nonetheless, sometimes the distribution is patchy, and cases of complete lateralization of the sensory block have been reported. Numerous radiologic studies have been carried out to exp;ain this patchy

anaesthetic distribution. Some studies have reported the presence of a median epidural septum, the plica mediana dorsalis. (Blomberg RG, Olsson S, 1989) Savolaine et al have described the presence of an anterior epidural space and dorsolateral epidural spaces due to the presence of the plica mediana dorsalis, which divides the posterior epidural space. Hogan has studied the epidural space using the cryomicrotome technique and did not find the median band but noted that the epidural space was not as homogenous as typically described.

In fact, ventrally, there is virtually no space between the duramater & the periosteum of the spinal canal; dorsally, the epidural space varies in size and can be described as a succession of small pyramidal space (at the level of ligamentum flava), which are separated from one another by strictures at the level of the vertebral laminae.

With regard to the contradictory opinions concerning the existence of the plica mediana dorsalis, attention should be paid to the nature of its constituting fibers. If these fibers are identical to those found everywhere in the epidural space with the only difference being their general orientation, one can imagine that the plica cannot be physiologically identified

Whatever the anatomic uncertainties, it should be remembered that the importance of the question to the anaesthesiologist is the space available for diffusion of the anaesthetic solution. After Harrisson, the epidural space can be considered a nerve or plexus sheath; any injection will result in distention of this sheath & the solution will spread following paths of least resistance. Usually, this diffusion of the solution is distributed evenly, but occasionally, some mechanical resistance forces, either physiologic, such as the plica mediana dorsalis, or pathologic, such as sequelae of epiduritis (following infection or surgery), can interfere, resulting in an incomplete sensory block Furthermore, if the speed of injection is too rapid, or the volume of solution is too large, the pressure within the subarachnoid space may become excessive leading

to neurological complications (Ramsey M, Roberts C 1991)

CONTENTS AND PRESSURE REGULATION IN THE EPIDURAL SPACE

The epidural space is filled with loose areolar tissue, fat, connective tissue fibers, blood vessels & lymphatics. The fat content of the epidural space is more in an obese individual, very fluid in infants, becoming more densely packed in children older than 7 to 8 years of age (Cheng PA 1963) and affecting the diffusion of the solution injected into the epidural space.

The epidural veins, which are numerous & organized in a plexus, communicate, on the one hand, with both the inferior vena cava and the azygous veins and on the other hand, via the cervical epidural veins and the circular sinus surrounding the foramen magnum, with the intracranial veins. As the epidural veins are valveless; they allow free passage of any solution or gas that may be accidentally injected.

At each spinal segment, the epidural space is traversed by a pair of spinal nerves, one on each side. At their origin, the spinal nerves are surrounded by extensions of the duramater, the so called "dural cuffs, which at times, are very thin. These dural cuffs present small invaginations - the arachnoid villi that protrude into the epidural space and are filled with cerebrospinal fluid. The epidural veins & lymphatics cross the duramater at the level of the dural cuffs and are responsible for a bidirectional exchange between the epidural and the subarachnoid spaces. Epuration and resorption of the cerebrospinal fluid occur at this level, and epidural solutions can use this passage to reach the subarachnoid space.

The presence of a negative pressure in the epidural space was first described by Jansen in 1926 & later by Heldt in 1928. At the thoracic level, the negative pressure in the epidural space is due to transmission of the negative intrathoracic pressure via the paravertebral spaces and the intervertebral foramina. At the lumbar level, the pressure is thought

to be due to displacement of the spinal dura by the tip of epidural needle. Changes in the blood flow in the epidural veins are in continuity with the inferior vena cava, any increase in the intraabdominal pressure - for example with coughing - could result in expansion of the epidural veins with a subsequent increase in epidural pressure.

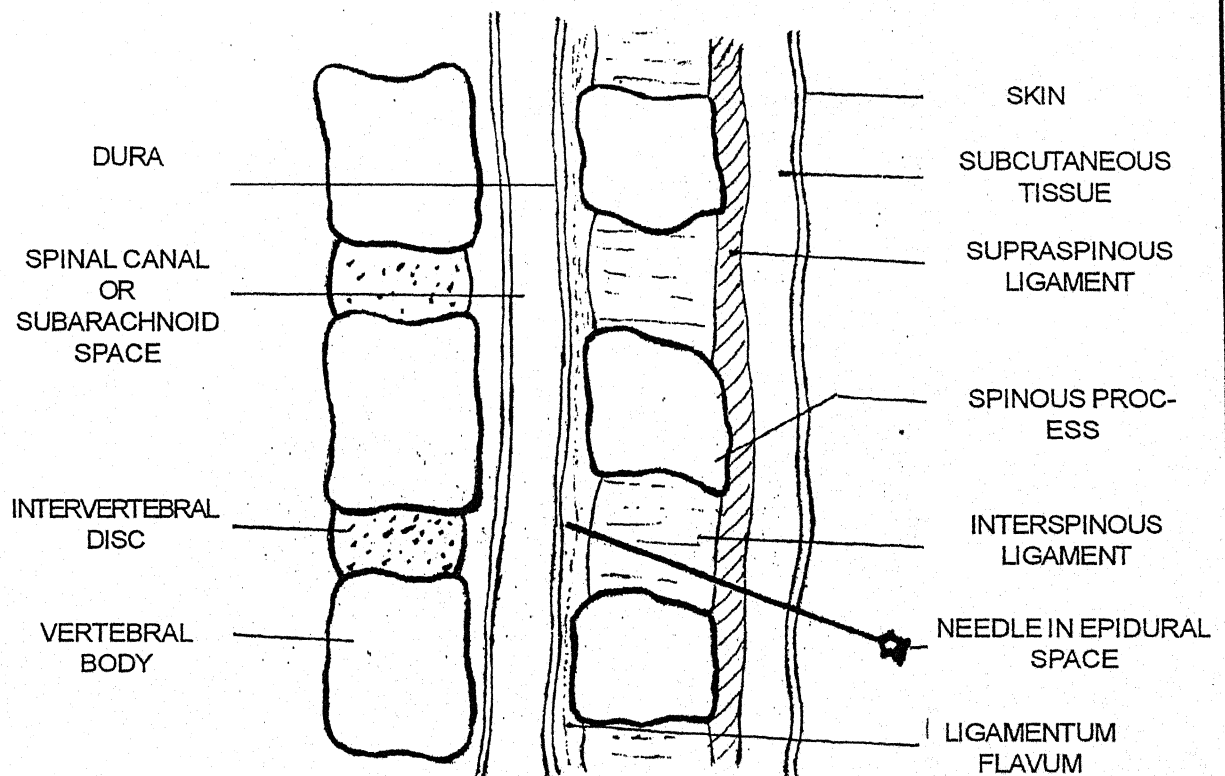
Andrade demonstrated the role played by the cerebrospinal fluid in the genesis of the negative epidural pressure. He suggested that the negative pressure of the epidural space could result from redistribution of the cerebrospinal fluid subsequent to changes in the position of the spine. Changing the position from the vertical to the horizontal plane decreases the hydrostatic pressure of the spinal subarachnoid space with subsequent changes in the shape of the dural sac. Because the vertebral canal is relatively stiff this change in shape would produce a suction effect which could result in a negative epidural pressure within minutes following the change in position.

ANATOMIC LANDMARKS OF THE EPIDURAL SPACE

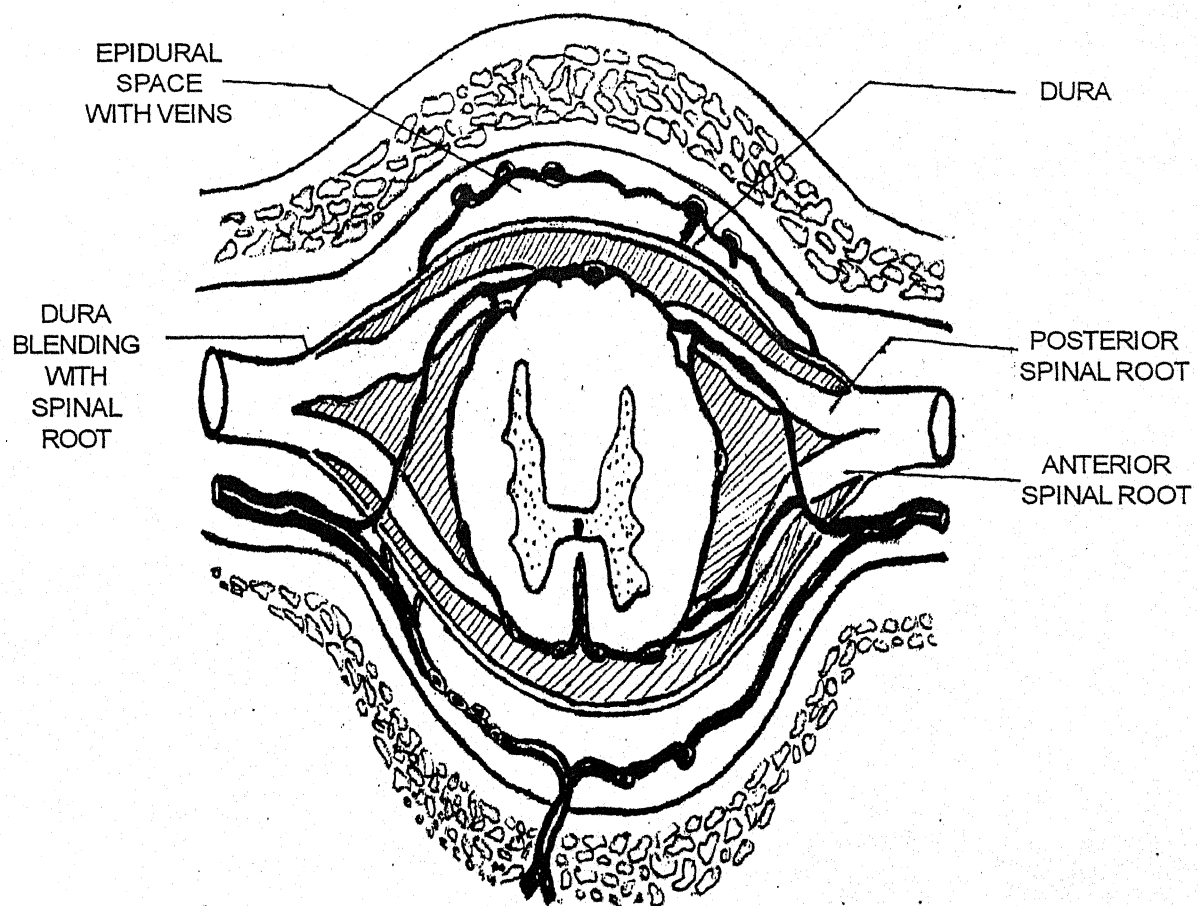
At the lumbar level, the line joining the two iliac crests crosses the upper border of the body of L₅ (L₄ in adolescents & adults). By palpating the spinous processes in the midline, it is easy to identify the desired intervertebral space.

Before entering the epidural space, the needle pierces the following structures :

- Skin and subcutaneous tissue.
- Supraspinous ligament with its fibers arranged longitudinally and firmly adherent to the vertebral spinous processes.
- Inter spinous ligament, which unites the inferior border of the upper spinous process with the superior border of the lower spinous process of two adjacent vertebrae; this ligament offers firm resistance to the epidural needle.



MIDLINE SECTION OF LUMBAR SPINE



DURAL CUFFS AND THE VENOUS PLEXUS OF THE EPIDURAL SPACE

- ✧ Ligamentum flavum, which unites the adjacent ipsilateral vertebral laminae; this is made up of elastic fibers that are densely packed; (Zarzur E, 1984) the ligamentum flavum becomes progressively thicker from the foramen magnum to the sacral hiatus; the sacrococcygeal membrane can be considered its caudal extension.

The distance between the skin and the epidural space depends on the age and weight of the patient. The structure of vertebrae plays an important role in determining the direction of needle. In the lumbar region where the spinous processes are almost horizontal and the interspinous ligaments are broader & thicker, the epidural needle can be advanced perpendicular to the interspinous line. The different tissue resistances are well felt and the chances of failed block are fewer, provided the lumbar spine is well flexed. At the midthoracic level, the spinous processes are markedly oblique; there is significant overlap between the adjacent vertebral laminae. Thus, to enter the epidural space at this level, the needle has to be directed obliquely in the cephalad direction. The epidural space can also be approached via a posterior paramedian or lateral approach. The needle traverses the skin, the subcutaneous tissue, the paravertebral muscles (which do not offer much resistance to the needle), remaining lateral to the supra and interspinous ligaments. If the needle is positioned correctly, it then lies on the ligamentum flavum in the midline and penetrates the epidural space laterally, where it is narrower. If the needle is more lateral, it would enter the ipsilateral paravertebral space, which is also accompanied by a loss of resistance, thus leading to an injection of the local anaesthetic in the wrong space and to failure of the block.

The epidural space surrounds the subarachnoid space. There are complex exchanges between, these two spaces especially at the level of the dural cuffs of the the spinal nerve roots. The relationship between these two spaces and their contents depend on the caliber of the spinal cord, particularly on its lumbar enlargement, which is made up of the

lumbar plexus nerves situated between L_3 & S_1 (the level depends on the age of the patient).

The curves of the vertebral column affect the width of the epidural space significantly. Lumbar lordosis brings the vertebral spinous processes together; this makes the lumbar approach to the epidural space difficult, so that, sometimes, it is not possible to approach the lumbar epidural space if the spine is not well flexed. The position of the patient also plays a very important role. (Jirout J, 1967). In the sitting position, the cord moves dorsally, reducing the safety margin of the epidural approach; however flexion of the lumbar spine neutralizes this. On the other hand, in the lateral position, the cord is displaced ventrally thus adding to the safety of the epidural approach. In the presence of spinal column deformities, such as kyphosis and scoliosis, the altered anatomy makes it difficult to approach the epidural space.

Because the spinal cord terminates at a lower level in children compared to adults, lumbar epidural anaesthesia is performed usually in the L_{4-5} or L_5S_1 ("Taylor approach) interspace in order to avoid direct trauma to the cord. In children older than age 10, a lumbar epidural can be performed at the L_{3-4} or L_{2-3} level.

The line joining the iliac crests usually crosses the interspinous line at the level of L_4 vertebral body. In fact the point where these two lines cross also varies with the degree of flexion of the spine and the age of the patient. In neonates, these lines cross above L_5 . In children above 5 to 6 years, it is not difficult to locate the correct interspace. In younger children, on the other hand, it may be easier to use the line joining the posterior inferior iliac spines since this line crosses the interspinous line at the body of S_2 . The desired interspace is then located by palpating and counting the spaces above this level.

SITE OF ACTION OF LOCAL ANAESTHETIC

The precise mechanism of the action of epidural anaesthesia still

remains controversial (Cousins MJ, Bromage PR, 1988). Probably, the mechanism of epidural anaesthesia is a result of complex interactions at the following sites :

- Spinal roots within their dural sleeves & arachnoid villi;
- Spinal ganglia:
- Periphery of the spinal cord via the subarachnoid space and the cerebrospinal fluid;
- Spinal nerves after the local anaesthetic solution passes into the paravertebral and perineural space through the intervertebral foramina.

SPREAD OF ANAESTHETIC SOLUTION

The spread of lumbar anaesthetic solution has not been extensively studied in children as the spread after a caudal injection. As in adults, the local anaesthetic solution spreads both cranially & caudally. The local anaesthetic solution tends to diffuse somewhat more in the cephalic direction, similar to the diffusion pattern in adult patients. Several studies have shown that a correlation exists between the segmental spread of anaesthesia and the age of the patient. (Bromage PR 1978; Ruston FG 1957). However, individual variations make it difficult to predict the exact level of anaesthesia. Factors affecting the spread of anaesthesia and the final level obtained for a given volume of anaesthetic solution in a given patient are several : the direction of the bevel of the needle, the speed of injection, and, also the position of the patient during and soon after the injection. Small volumes of the injection solution made with the patient in the lateral position increase the chances of lateralization of the epidural. The assessment of the height of the block also depends on the method used to verify the motor, sensory and sympathetic block. (Cousins MJ, Bromage PR 1988) Pinching the skin has been shown to have the best anatomic, radiologic, and clinical correlation.

METHODS OF IDENTIFYING THE EPIDURAL SPACE

Epidural anaesthesia requires a procedure for precise identification of the posterior epidural space as the needle passes through the ligamentum flavum (Bromage PR, 1978). Several methods for detecting the epidural space have been described. Ruston, in 1957, described the "hanging drop technique" which was used initially for caudal anaesthesia.

In the "Hanging drop" method the epidural needle is gradually advanced in the patient's back with a drop of normal saline or fluid hanging from its hub. As the tip of the needle enters the epidural space, the drop is sucked into the epidural space, due to the negative pressure. One can then inject the local anaesthetic solution while respecting the safety measures. The technique is still used for thoracic epidural performed in the sitting position but not for the lumbar epidural. Performing this technique in neonates in the lateral decubitus is not easy as the negative pressure in epidural space is very slight or sometimes absent. Modifications of this technique eg; the Macintosh balloon, have been described but are used infrequently.

The most commonly used technique for the detection of the posterior epidural space is the loss of resistance technique. A syringe filled with 1 to 3 ml of normal saline solution, air or medical CO_2 is attached to the epidural needle. The needle is advanced into the epidural space by maintaining a firm pressure on the piston of the syringe, if normal saline is used, or using pulsatile pressure with the thumb, if gas is used, to detect the loss of resistance. As the tip of needle enters the epidural space there is a sudden loss of resistance and piston moves freely forwards in the syringe.

The syringe should have a piston that moves freely, without friction, and without leakage, thereby allowing perception of very fine change in tissue resistance as the needle advances. Two types of syringes meet these criteria. The first is the glass syringe whose piston is initially immersed in the local anaesthetic solution, thus acting as a

lubricant and providing a tighter seal . Recently , plastic syringes have been developed especially for this purpose. These latter syringes are very reliable even in neonates.

Loss of resistance using fluid is used more commonly in adults but also in paediatric patients. Fluid can dilute the concentration of the local anaesthetic solution, especially in neonates where the total volume of injection of the local anaesthetic is rather small (Arthur DS, Mc Nicol LR 1986). This complicates somewhat the identification of a puncture of the dura mater, because the fluid can drip back through the needle, simulating a reflux of cerebrospinal fluid. The difference between the two can be distinguished by the characteristic flow of the cerebrospinal fluid. In doubtful cases, cerebrospinal fluid can be ruled out by documenting the absence of glucose.

Detection of loss of resistance using air is more reliable especially in neonates and infants whose tissues are less densely packed and more hydrated than those of older patients. Using nonsterile ambient air is not without risks, especially if a large volume of air is injected. This can give rise to several complications of which air embolism, compressive cervical emphysema, spinal compression leading to paraplegia (reversed by surgical correction) or, less importantly, epidural bubbles, which may interfere with the action of the local anaesthetic solution have been reported. (Dalens B, Bazin JE, Haberer J.P. 1987) The air can persist in the epidural space for several weeks after the procedure, especially if nitrous oxide has been used in the course of anaesthesia. Dalens recommends the loss of resistance technique using medical CO₂ in small volumes (Maximum 4 ml) because it is sterile and easily absorbed. (Dalens B, 1991).

VOLUME OF INJECTION:

The volume to be injected depends on the number of segments that one desires to block. Schulte- Steinberg put forward a formulae,

which states that the volume of local anaesthetic required to block one spinal segment is equal to one tenth the age in years (upto puberty) i.e.

$$V(\text{ML/ neuromere}) = 1/10 \times \text{age (in years)}$$

In practice, 0.5 to 1.0 ml / kg upto a maximum of 20 ml is injected slowly. This usually produces analgesia between T4 and T12 and, on an average, T9 to T10 if 1 ml /kg is used.

POSITION OF THE PATIENT:

SITTING POSITION:-

This position is used frequently in adult patients. It can be used in children who are awake and cooperative, especially in those over the age of 7 to 8 years. The patient is made to sit at the edge of the table with the legs hanging; the abdomen is supported with a pillow or a folded blanket. An assistant stands in front of the patient to make sure that the back remains flexed in order to widen the interspinous spaces. Although this position increases the ease of the epidural puncture, it also marginally increases the risk of an inadvertent dural puncture because the spinal cord moves dorsally with the patient in the sitting position, and the pressure in the cerebrospinal fluid increases.

LATERAL DECUBITUS:-

This position is more commonly used in paediatric patient, especially those under anaesthesia. The patient is made to lie on the side that is being operated on; the legs are flexed, and the head is supported with a pillow, the lower arm is placed perpendicular to the body and the upper arm is laid across the chest. When the size of the pillow is correct the line of the spinous processes is practically parallel to the operating table. The pelvis does not play as important a role in children as in adults, and even in adolescent patients, it is rare that the line joining the interspinous processes is very oblique in the rostrocaudal sense as it is in adult female.

PRONE POSITION:-

This position is very rarely used. It may be used in patients who have a large plaster cast and in patients with spinal deformities in which a lateral decubitus or a sitting position is not possible. The prone position tends to accentuate the lumbar lordosis, thereby making epidural access difficult. The lordosis is countered by placing a rolled towel under each anterior superior iliac spine or by placing a patient on a frame used for Scoliosis surgery (Relton- Hall Frame).

TECHNIQUE:-

SINGLE SHOT MEDIAN LUMBAR EPIDURAL :-

The patient is appropriately positioned; the site of puncture is marked and cleaned thoroughly with an antiseptic solution. Sterile drapes are placed. The skin is punctured either directly with the epidural needle or using a breaking needle. The epidural needle with stylet is introduced with the bevel parallel to the sagittal plane through the skin and subcutaneous tissue and perpendicular to the interspinous line. Once the needle enters the inter spinous ligament (felt as an increase in resistance) the stylet is withdrawn & a syringe (for detecting loss of resistance) is attached. The epidural needle is then slowly advanced in the same plane with a continuous pressure (fluid detection) or tremolo (air detection) on the piston of the syringe throughout the procedure.

In order to avoid gross penetration by the needle, the needle is stabilized with the anaesthesiologist's free hand. This provides counterpressure to the advancing hand. The needle encounters increasing resistance accompanied by characteristic "cracks" as it advances through the interspinous ligament. The at least in children older than 3 years of age- resistance offered to the needle increases considerably as it pierces the ligamentum flavum. Shortly after crossing the ligamentum flavum, there is a sudden loss of resistance when the piston of the syringe enters the barrel freely. This indicates that the tip of the needle is in the posterior epidural space. The distance from the skin

to the epidural space varies with the age and weight of the patient. If the spine is not adequately flexed or if the needle is not directed perpendicularly, it may hit against the vertebral lamina. If the needle encounters bone, the needle should be retracted and redirected after confirming the position of the patient and the anatomic landmarks.

Once the needle is in epidural space; its position is fixed and the bevel is directed in the cephalad direction. The syringe is disconnected, and the hub of the needle is carefully scrutinized for reflux of blood or any other fluid. If the fluid is used for detecting a loss of resistance, a few drops of fluid may drip from the hub of needle, which should then stop immediately. If leakage persists or if, there is any suspicion that the dura has been punctured, one should confirm the absence of glucose using Dextrostix. If the Dextrostix is positive for glucose, this indicates that the fluid is cerebrospinal fluid ie; that there is an inadvertent dural puncture. The needle is then withdrawn, and the procedure attempted in the intervertebral space immediately above the present space. If a vessel is punctured, the needle should be withdrawn, and a second epidural attempted in the same (or another) intervertebral space.

In the absence of any reflux, the syringe used to detect the loss of resistance is reconnected to the needle and aspirated. If the aspiration test is negative (i.e. there is no reflux of fluid or blood) the syringe containing the local anaesthetic solution is attached to the needle. After a negative aspiration test, a test dose of 0.5 to 1 ml that contains epinephrine is made with continuous monitoring of the heart rate and blood pressure. If no change in heart rate or arrhythmia is observed over a period of 45-60 second, the local anaesthetic solution is then injected slowly. The syringe is repeatedly aspirated during the course of the injection.

The speed of injection is a matter of debate (Dalens B, Haberer JP 1987). A rapid injection may raise the level of analgesia and also increase the epidural and subarachnoid pressures. A slow injection may increase

the possibility of lateralization of the epidural, especially in the lateral decubitus position. It would also result in lowering the upper level of block which, therefore would necessitate an additional dose and hence, increase the risk of toxicity. In practice, it is advisable to inject the solution over a period of 90-120 seconds, irrespective of the age of the patient and the volume used.

Due to the respiratory movements, slight movement of the epidural needle occurs during the procedure. These movements can be minimized by interposing a tubing between the needle and the syringe as suggested by Winnie for the peripheral nerve blocks. The tubing must be purged with local anaesthetic solution prior to attaching it to the needle. The interposed tubing reduces the chances of secondary displacement of the needle during the course of injection because the movements of the syringe are not transmitted to the needle.

SINGLE SHOT PARAMEDIAN LUMBAR EPIDURAL

Paramedian (also known as paravertebral or lateral) lumbar epidural anaesthesia is rarely used in the paediatric patients. It is indicated only if the midline approach fails or in the presence of spinal deformities, when adequate flexion of the spine cannot be achieved. At each intervertebral level, there are two symmetrical puncture points. The point that is most suitable is chosen. The epidural needle with its stylet is introduced in the horizontal plane with the needle directed medially at an angle of 15° to 20° to the ligamentum flavum. After passing through the subcutaneous tissue, the needle passes through the paravertebral muscles, which are more difficult to identify than the interspinous ligaments encountered in the median approach. The stylet is then withdrawn; the syringe used to detect a loss of resistance is attached & the needle is then advanced with a continuous pressure (fluid detection) or tremolo (gas detection) on the piston of the syringe. The forward movement of the needle must be controlled by the index finger of the left hand of the anaesthesiologist.

As the needle enters the ligamentum flavum, an increase in resistance is felt. The needle is then cautiously advanced by 1 to 3 mm until a sudden loss of resistance is felt, indicating that the tip of the needle is in the epidural space. The distance at which the epidural space is detected is almost similar to that using the median approach at that particular level. Once the needle is in the epidural space the same procedure as that of a median approach is followed. The needle may hit against a bony structure if the angle is not maintained. Sometimes even if the angle is right, it can touch a vertebral lamina. If this happens, the needle should be withdrawn and the procedure started from the beginning. If the medial angle is more than 25° , then the needle will not enter the ligamentum flavum but will enter the contralateral paravertebral space. If the medial angle is lesser than 15° then the needle can enter the ipsilateral paravertebral space. If the local anaesthetic solution is injected here, it will create a paravertebral block & not an epidural block. If the flexion of lumbar spine is inadequate, it may not be possible to enter the epidural space using the above technique. In such a case, it is advisable to use the technique described by Cousins and Bromage. Using this Method the puncture point is much lower - almost at the level of the spinous process of the lower vertebra. The needle is then directed not just at an angle of 15° to 20° medially but also upward, at an angle of 45° . The rest of the procedure is similar to a typical paramedian approach.

CONTRAINDICATIONS.

Absolute contraindications to a lumbar epidural anaesthetic procedure include :

- Coagulation disorders
- Septicemia
- Meningitis
- Infection or dystrophic lesion at the puncture site
- Uncorrected hypovolemia

➤ Allergy to local anaesthetics.

Progressive degenerative neuropathy usually carries with it medicolegal implications and is, thus, a relative contra-indication, although there has been no evidence to show that an epidural anaesthetic would be harmful in such patients. Patients on anticoagulant medications also pose problems; several studies have been carried out which are contradictory. Although certain studies have shown that there is no increase in morbidity in patients who are on anticoagulant solutions it would be advisable to avoid epidurals in patients who are on therapeutic anticoagulation. If prophylactic anticoagulant therapy is initiated in the post operative period, most authors believe that this should not significantly increase morbidity if coagulation is satisfactory at the time of epidural puncture. Although research is still insufficient on this point at present, administration of low molecular weight heparins may not preclude their use with epidurals in future.

Anatomic malformations of the spine, such as spina bifida, meningocele, and intracranial tumors are also absolute contraindications, even though a recent study has described use of epidural analgesia in patients with congenital lumbosacral spinal anomalies. Hydrocephalus and a reduction of the intracranial compliance, as in craniosynostosis, are also considered absolute contraindications by many authors. (Dalens B; Hilt H, Link J; Usubiaga JE. Maya F, Usubiaga LE; Wildsmith JAW, 1967, 1986)

In the presence of diastematomyelia, the decision on epidural anaesthesia is not easy. In the absence of neurological symptoms or the spinal deformity associated with it, this malformation is often unrecognised, and, according to all evidence, numerous epidural anaesthesias have been carried out in this area without anyone having described significant incidence of particularly important complications. According to Dalens, diastematomyelia, does not constitute, an absolute contraindication, even if a minor neurological symptomatology should

exist.

Presence of hemivertebrae, kyphosis, scoliosis & previous surgery of the spine are not contraindications for an epidural. However, these deformities can make it difficult & even impossible, to perform the epidural. Moreover, the risks involved are sometimes considerable because it is often impossible to control the movements of the spinal cord within the spinal canal. Finally, if surgery has been performed on spine, intervertebral access may be difficult, or even if the needle reaches the epidural space easily, the spread of the local anaesthetic may be uneven because of adhesions, thus resulting in patchy anaesthesia. For this reason, it would be wise to avoid epidural anaesthesia in these patients.

Epilepsy or a history of convulsions are considered contraindications to an epidural by certain authors, although there are no data to support this. In fact, the local anaesthetic solutions, due to their inherent anticonvulsant properties may be of some benefit to these patients. There is no objective evidence for precluding epidural anaesthesia in epileptics; the only drawback is the likelihood that epidural anaesthesia would be blamed if convulsions were to occur in such patients.

SIDE EFFECTS AND COMPLICATIONS :

I - COMPLICATIONS OF PUNCTURE :

1) SUBCUTANEOUS AND INTRAMUSCULAR INJECTION :

In the neonate, the high degree of hydration & the low density of the tissues reduce the characteristic resistance and one feels a "Give" similar to a loss of resistance as the needle enters the subcutaneous tissue just before puncturing the supraspinous ligament in the midline approach or when the needle penetrates the paravertebral muscles at the lateral paramedian approach. One can avoid the false localization of the epidural space by recalling the distance calculated for that epidural space & expected for that particular patient as determined by patient's

age and weight. If one injects the local anaesthetic solution subcutaneously or intramuscularly, it would lead simply to failure of the technique because there would be no analgesic effect.

2) INJECTION INTO THE PARAVERTEBRAL SPACE:-

When the needle enters the paravertebral space, the loss of resistance felt is similar to that when the epidural space is entered. It is usually seen when the lateral approach to the epidural space is used; one should suspect it when there is loss of resistance felt without a previous increase in resistance. An injection into the paravertebral space gives rise to a paravertebral block wherein a single nerve root is blocked at that particular level. Because the epidural space communicates freely with the paravertebral space some of the anaesthetic solution injected in the paravertebral space may enter the epidural space and may give rise to somewhat patchy anaesthetic effect, which may then be misinterpreted as secondary to a partial epidural block despite a satisfactory technique, as that due to adhesions or septa in the epidural space.

3) INJURY TO BLOOD VESSELS & PARAPLEGIA

Introducing a needle into a vascular space like the epidural space can lead to vessel injury. Inadvertent vessel puncture is usually without any consequences provided that it is detected prior to injection of a local anaesthetic. Epidural hematomas leading to compression of spinal cord have been reported and almost always seen in patients on anticoagulant therapy (Cousins MJ, 1972)

Ischaemic complications leading to paraplegia have been reported mainly following a spinal anaesthetic and sometimes following an epidural. Sphincter control & sensations usually return in a few days or a few months; however, motor impairment is usually permanent. The factors responsible for this may be severe vasoconstriction due to the use of epinephrine, severe uncorrected hypotension in adults, & poor venous

return.

4) DURAL PUNCTURE

Dural puncture may be seen when inappropriate equipment is used, if the loss of resistance technique is not used correctly or when there is poor control of needle penetration into the tissues. The diagnosis of dural puncture can be made when clear fluid is seen to flow through the needle hub either spontaneously or after aspiration, and the fluid tests positive for glucose. The needle should be immediately withdrawn & a second epidural can be attempted in the intervertebral space just above.

The consequences of dural puncture are negligible in pediatric patients. Post dural puncture headache is exceptional before the age of 10 years & is unusual before puberty. Headache that persists 72 hours following bed rest should be treated by a blood patch.

5) DAMAGE TO THE SPINAL CORD OR NERVE ROOTS:-

In case of lumbar epidural above L₃, the needle can tear a nerve root of the cauda equina or damage the conus medullaris itself if the movement of the needle is not well controlled. This may give rise to some sensory or motor neurological deficits. The sensory deficits may be persistent, whereas the motor deficit may resolve over a few days. Cauda equina and paraplegia may occur.

These complications result from a combination of factors & rarely due to trauma alone. Such factors include vascular injury & severe uncorrected hypotension. An epidural hematoma, especially at the point of entry of the spinal artery in the spinal cord, can compress the nerve roots & also cause ischaemic damage to the cord if it compresses a spinal artery. Severe hypotension is rarely seen in paediatric patients.

An intraneural injection of local anaesthetic due to direct trauma of a nerve root by the needle can cause permanent damage to the nerve fibers.

COMPLICATIONS & TOXICITY RELATED TO THE INJECTED SOLUTION:-

1) INTRAVASCULAR INJECTION:

The dense venous plexus of the epidural space increases the risk of vascular puncture by the epidural needle or by the tip of the epidural catheter. Reflux of blood, either spontaneous or after aspiration, is usually, but not always, seen. If it goes undetected an inadvertent intravascular injection may result in systemic toxicity.

2) SUBARACHNOID INJECTION:

Undetected subarachnoid puncture with subsequent injection of the local anaesthetic solution intended for an epidural injection, leads to a total spinal anaesthesia (Hodgkinson R. 1981). Within seconds of the injection, the patient will complain of difficulty in breathing followed by apnoea. If appropriately treated with controlled ventilation and the use of sympathomimetics, the respiratory function and sympathetic tone are restored within 60-90 minutes without any further sequelae.

If the injection is subdural, there is a latent period between injection and the onset of spinal block, and the injection may not be associated with a respiratory arrest or motor block.

3) SUBDURAL INJECTION:

Subdural injection, as a complication associated with an epidural injection, is difficult to avoid (Sechzer PH, 1963). An apparent epidural injection, which from a technical point of view has gone smoothly, may be followed by an extensive sensory block out of proportion to the dose of local anaesthetic injected; the cranial nerves up to the trigeminal nerve can be involved. In comparison, the motor and sympathetic block is minimal. It appears within 20 minutes after the injection and lasts for about 60 minutes.

4) HEMODYNAMIC ALTERATIONS:

Hypotension is usually not seen in children (Murat I, Egu JF. 1987). even if associated with an inadvertent total spinal injection. After 8 years of age, a 20% drop in blood pressure compared to preoperative values is seen in 5% of the children but rarely requires treatment with sympathomimetic solution or volume overloading. Preloading is not required in a child because the volume of blood that stagnates in the lower extremities is not significant (compared to adults where the amount can be as much as 600 ml).

5) PARTIAL OR COMPLETE FAILURE OF THE BLOCK :-

Complete failure of the block is usually seen if the epidural space is wrongly identified and the injection is made either in the vertebral muscles or the paravertebral space. The latter would lead to a paravertebral block where only a single spinal nerve would be blocked.

6) LATERALIZATION OF THE BLOCK

Frequently there is a difference of as much as two spinal segments in the level of the anaesthetized area on opposite side of the midline, esp. if the injection is made with the patient in the lateral decubitus position. The difference may be upto 4 dermatomes, particularly in a neonate, if the local anaesthetic has been injected very slowly. Complete lateralization of the epidural may occur because of the presence of adhesions that have developed following surgery, or it may be due to infection or inflammation. However, most often, complete lateralization is due to the presence of a complete plica mediana dorsalis, which divides the posterior epidural space in to two halves. (Bailey P.W., 1986)

7) UNANESTHETISED DERMATOMES:

Rarely, the anaesthetic effect may be uneven and unanesthetised areas may persist (Bromage PR, 1972). When it occurs, it is more frequently seen at the S1-S2 level and has been attributed to poor penetration of the local anaesthetic solution into the large spinal roots at this level.

The use of air for detection of the epidural space by the loss of resistance technique has also been implicated. (Dalens B & others 1987). Air contains a high concentration of nitrogen which is difficult to eliminate not only from the epidural space but also from the human body in general. Thus, air when injected into the epidural space, can form persistent bubbles around the nerve roots thereby preventing the local anaesthetic from coming in contact with them. Medical CO₂, on the other hand, is highly diffusible & would not give rise to unevenness of the anaesthetic distribution. Other complications from the use of air are air embolism, cervical emphysema, and spinal compression.

8) INAPPROPRIATE HEIGHT OF THE BLOCK :-

If the volume and conc. of local anaesthetic are inadequate one may have a lower level of block, necessitating the use of other complementary analgesics. On the other hand, if a large volume and concentration of the local anaesthetic are used, one will get a higher level of block with subsequent respiratory problems. (Defalque RJ, 1967) A slowly developing block that lasts for a prolonged period should make one suspect a subdural injection.

OTHER COMPLICATIONS :

1) SENSORY DISTURBANCES AND LOSS OF CONSCIOUSNESS :

Minor sensory disturbances and problems of sphincter control are not usually observed in children. Transient involvement of the cranial nerves with the exception of the 1st, 9th & 10th pairs has been reported. This involvement may be due to the subdural injection of the local anaesthetic solution.

2) INFECTION AND CHEMICAL MENINGITIS :

Complications, such as epidural abscess, meningitis & discitis, have been relatively frequent in cases where needles have been reused.

3) DERMOID CYSTS :

Introduction of epidermal cells in the spinal canal increases the chances of an intraspinal dermoid tumor (Batnitzky S & others 1977) which can lead to cord compression or cauda equina syndrome that may require surgical excision. It is rare in practice and can be avoided if a needle with stylet is used or if a breaking needle has been used prior to the insertion of the epidural needle.

4) POOR PSYCHOLOGICAL TOLERANCE OF THE SECONDARY EFFECTS OF THE BLOCK :

A child between the age of 3-6 yrs may not be able to tolerate the presence of a motor or a sensory block during the recovery period. This situation can be avoided if the procedure has been explained to the child and to the parents in detail during the preoperative visit.

5) FLARING OF THE LATENT INFECTION :

Epidural anaesthesia has been implicated in reactivating infections, for example herpes. Of greater concern is the risk of unmasking latent neurologic diseases, such as, spinal cord compression, a cerebral tumor, an angioma, or an epidural abscess.

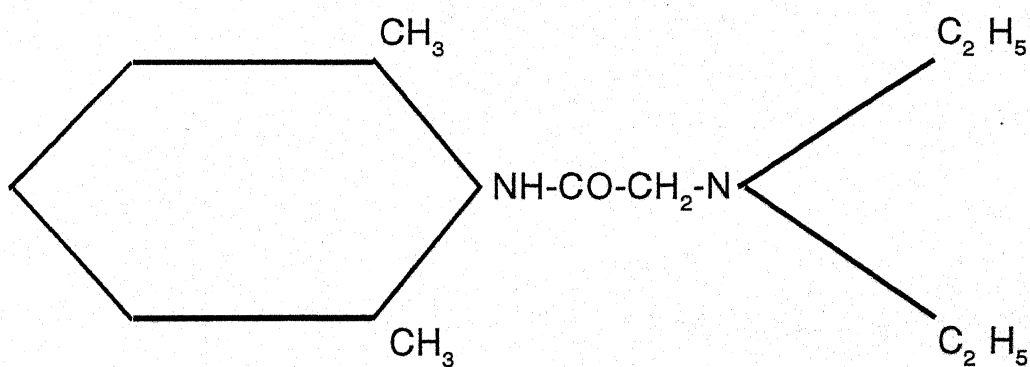
6) ALLERGY TO LOCAL ANAESTHETICS :

True allergy to local anaesthetics is extremely rare and is usually seen with aminoesters.

7) SHIVERING :

If the temperature of the injected solution is lower than 37° C, it may stimulate temperature receptors in the epidural space. In order to avoid or reduce the incidence of shivering, the local anaesthetic solution should be warmed to body temperature prior to injecting it in the epidural space.

Lignocaine (2 diethylamino 2, 6-acetoxylidide)



STRUCTURAL FORMULA

STRUCTURAL FORMULA

It was synthesised by Nils Iofgren (1922-1953) and Lundqvist in 1943 in Sweden. First used by Gordh (1907) of the Karolinska Hospital, Stockholm in 1948.

CHEMISTRY :

Lignocaine belongs to amide group of local anaesthetics and has a basic structure termed tertiary amide. It is very stable not decomposed by boiling, acids or alkalis. The pKa is 7.86. It occurs as colourless crystals, freely soluble as the hydrochloride salt in water, melting at 66.69° C.

CLINICAL PHARMACOLOGY :

MECHANISM OF ACTION :

Lignocaine stabilizes the neuronal membrane by inhibiting ionic fluxes required for initiation and conduction of impulses thereby effecting local anaesthetic action.

PHARMACOKINETICS AND METABOLISM :

Lignocaine is completely absorbed following parenteral administration, its rate of absorption depending upon various factors such as site of administration and the presence or absence of vasoconstrictor agent. Except for intravascular injection, the highest blood levels are obtained following intercostal nerve block and the lowest after subcutaneous injection. The plasma binding (mainly with alpha-1 acid glycoprotein) of lignocaine is dependent on drug concentration, and the fraction bound decreases with increasing concentration. Lignocaine crosses the blood brain and placental barriers, presumably by passive diffusion.

Lignocaine is metabolised rapidly by the liver, and metabolites are excreted by the kidneys. Approximately 90% of lignocaine administered is excreted in the form of various metabolites and less than 10% is excreted

unchanged. The primary metabolite in urine is a conjugate of 4 hydroxy 2, 6 dimethylaniline. It is neither a vasodilator nor does it interfere with the vasoconstrictive action of adrenaline. It is metabolized by oxidases and amidases from microsomes in the liver but this is retarded in chronic liver disease.

The elimination half life of lignocaine following an intravenous bolus injection is typically 1.5 to 2 hours. The half life may be prolonged in patients with liver dysfunction. Renal dysfunction does not affect lignocaine kinetics but increase the accumulation of metabolites.

Factors such as acidosis and the use of CNS stimulants and depressants affect the CNS levels of lignocaine required to produce overt systemic effects.

SYSTEMIC EFFECT :

These effects occur either due to systemic absorption or due to accidental intravenous injection. The chief systemic effects are on the cardiovascular system and central nervous system.

CARDIOVASCULAR SYSTEM :

THE HEART :

Lignocaine has a stabilising effect on the cell membrane of cardiac tissue. It tends to depress automaticity in abnormal or damaged fibers and thereby suppress cardiac dysrhythmias. Lignocaine is remarkably non cardiotoxic when used clinically.

VASCULAR SMOOTH MUSCLE :

The regional effect is simply vasodilatation in the area supplied by blocked sympathetic nerves. Systemic effects may be produced in a variety of ways, reflexly and because of central nervous system involvement, but not as a result of any direct action on blood vessels, because the ambient concentration would be too low. Effects on the circulation of subconvulsive doses are variable but minimal, while large

doses may produce circulatory collapse as a result of medullary depression and of convulsions causing respiratory impairment, rather than because of any direct effect on the circulation. The first signs of local anaesthetic toxicity in a pediatric patient may be dysrhythmias or cardiovascular collapse.

CENTRAL NERVOUS SYSTEM :

Sedation and lightheadedness are seen. With more marked toxicity the patient may notice a numb tongue, circumoral pins and needles, twitching and visual disturbances. Severe toxicity proceeds to convulsions and coma with respiratory and cardiac depression, as a result of medullary depression.

SOME IMPORTANT POINTS REGARDING LOCAL ANAESTHETIC IN RELATION TO CHILDREN

1) Local anaesthetics bind to plasma albumin but the principal binding plasma protein is alpha alpha 1- acid glycoprotein.

(Denson DD, Coyle DE, Thompson GA, Myers JA: 1984, WOOD M; 1986)

The free (not bound to plasma proteins) amount of local anaesthetic is higher in the neonate due to the lower levels of alpha -1-acid glycoprotein. The plasma concentration of alpha -1 acid glycoprotein increases gradually with age, reaching adult level at 6 months of age.

The free fraction of lidocaine is twice as high (48%) in the newborn as compared to an infant or adult (26%) (Lerman J, Strong HA, Le Dez KM, Burrows F.A. 1989)

Due to its weaker protein binding, lesser influence of age on the free plasma fraction and lesser systemic toxicity, lidocaine is preferred to bupivacaine prior to 6 months of age.

2) The protein binding of lidocaine is the same in infants whether or not they have cyanotic heart disease. (Burrows FA, Lerman J, LeDez KM, Strong A; 1990)

3) Nerve conduction in nonmyelinated nerve fibers is similar in both adults and children, whereas, in myelinated nerve fibers, nerve conduction is slower in children. (Benzon HT, Strichartz GR, Gissen AJ, Shanks CA, Covino BG, Datta S; 1988).

In an infant because the fiber diameter is smaller, the myelin sheath thinner, and the internodal distance smaller, the C_m (minimum anaesthetic concentration) of the local anaesthetics is less, irrespective of the type of block.

4) The large volumes of local anaesthetic used in children coupled with the shorter length of the nerve fibers explains why good quality of

anaesthesia is achieved despite dilute concentrations.

5) Local spread is easier in children than in adults, especially in the epidural space because epidural fat is dense in adults, but sparse & less dense in children.

(Bosenberg AT, Bland BAR, Schulte-Steinberg O, Downing JW; 1988)

6) Following an epidural injection, the classic biphasic absorption curve, which is related to the epidural fat; is not seen in children & thus the absorption of local anaesthetic is much more rapid in the latter compared to adults.

7) Cardiac output & regional blood flow, corrected for weight is greater in young children compared to older children and adults. Thus, whatever be the site of injection, systemic absorption of the local anaesthetic will be greater in young children.

(Eyres RL; Bishop W; Oppenheim RC; Brown TCK; Hastings; 1983, 1986)

8) Lower levels of plasma alpha -1-acid glycoprotein result in a higher free fraction of the local anaesthetic in the newborn which in turn; increases the risk of toxicity.

9) Extracellular fluid decreases with increasing age and eventually reaches the adult value of 20-25% of body weight at 2 years of age. Intracellular fluid, on the other hand, increases gradually, and by the end of second year of life reaches the adult value of 40% of the body weight. Consequently, the volume of distribution of the local anaesthetic is greater at birth and decreases progressively with increasing age.

10) In children, due to the rapid rate of absorption, the plasma level is very high. This is somewhat offset by the relative increase in the volume of distribution of the local anaesthetic in the newborn & the infant.

11) Most of the microsomal enzymes required for the biotrans

formation of the aminoamides are functional right after birth. However their activity is considered weaker in newborns compared to adults. This explains the pharmacokinetic differences between the newborn and the adult. (Difazio CA, 1979).

12) The phase II reaction, in particular glucuronidation, is immature right from birth up to the age of 3 years. On the other hand, the biotransformation of lidocaine depends mainly on the hepatic blood flow and very little on the enzymatic processes. Thus, the plasma clearance of lidocaine does not differ in the newborn and the adult. In children, over the age of 1 year, the plasma clearance of the local anaesthetics is greater compared to adults as the cardiac output and regional blood flow are higher in the former.

13) In general, the elimination half life of the local anaesthetics differs very little in the children older than 1 year and in adults. In fact, the increase in the volume of distribution is compensated for by an increase in the plasma clearance, the latter resulting from the relatively greater hepatic blood flow in children - the liver representing 4% of body weight in children as opposed to 2% in adults.

In the neonate, the decreased clearance occurs due to the prolonged elimination half life of the local anaesthetics, which would increase the risk of cumulative toxicity following repeated injections. However, a study has shown very little difference between the newborn or the infant and adult.

(Bricker SRW, Telford RJ, Booker P.D. 1989)

14) In paediatrics, regional anaesthesia is often combined with a light general anaesthesia. This raises the threshold of toxicity of the local anaesthetics. Thus the dose of lidocaine, required to induce convulsions in animals is 22 mg/kg in an anaesthetized animal as compared to 5.8 mg/kg in a conscious animal.

➤ (Liu P, Feldman HS, Covino BM, Giasi R, Covino B.G. 1982)

✧ (Morishima HO, Pedersen H, Finster M, Sakuma K, Bruce SL; 1981)

✧ (Munson E.S. 1975)

15) The immaturity of central nervous system probably plays a role in protecting against neurotoxicity of local anaesthetic agent.

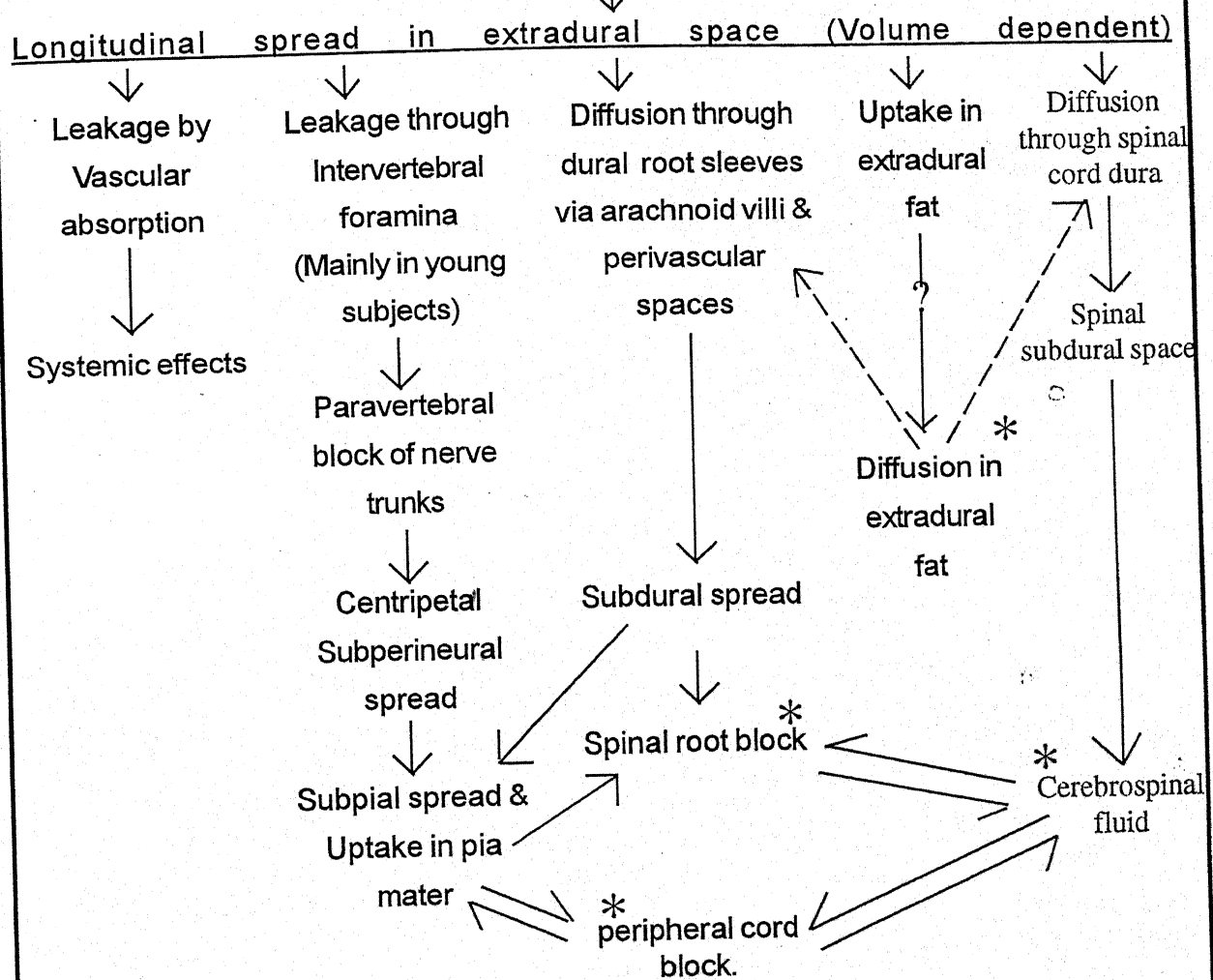
Neurotoxicity of lidocaine is increased in cases of right to left cardiac shunts.

(Bokesch PM, Castaneda AR, Ziemer G, Wilson JM; 1987).

16) Hypersensitivity to local anaesthetic is rare in paediatrics. Ester derivatives of PABA have been responsible for most reports of hypersensitivity while true allergy to the aminoamides is extremely rare.

THE FATE OF AN EPIDURAL INJECTION

EXTRADURAL INJECTION



* Sites where experimental measurement has demonstrated local anaesthetic concentrations in excess of the minimal effective concentration (C_m).

MATERIAL AND METHOD

MATERIAL AND METHOD

The present study was carried out on sixty paediatric patients admitted in surgical and orthopaedic units of M.L.B. Medical college and Hosptial Jhansi, during the year 1996-1997.

SUBJECT OF STUDY:-

The aim of the study was to see the efficiacy, safety and cardiorespiratory stability under epidural anaesthesia in children undergoing various lower abdominal and lower limb surgical interventions:-

MATERIAL:

The material comprised of-

- 21 G spinal needle
- Glass syringes
- Pulse oximeter for the measurement of oxygen saturation.
- Sphygnomanometer with paediatric size cuff for blood pressure measurement.
- Local anaesthetic- Lignocaine hydrochloride with adrenaline.

SELECTION OF PATIENTS:-

The patients of either sex selected for study were those kept for operation by the department of surgery and orthopaedics as a routine or emergency case. The patients selected were of ASA Grade I and Grade II, between the age group of 1 to 12 years, undergoing lower abdominal, perineal or lower limb surgery.

Patients name, age, sex, Weight and M.R.D. number were noted and a thorough history and physical examination was done. The patients who were excluded from the study were-

- Patients with respiratory disorder
- Patients with cardiovascular disorder

- Patients with neurological disorder
- Patients with disease of spine
- patients with skin lesion at the site of lumbar puncture.
- Patients with any bleeding disorder.

The patients were advised to undergo following investigations:-

- Hb, TLC, DLC
- Routine and microscopic examination of urine.
- Bleeding time
- Clotting time.
- Other specific tests were advised as and when indicated.

All the procedures and possible risks & complications were explained to the patient's attendants & informed consent was obtained. Sensitivity test for lignocaine preceded the procedure.

The total span of Work comprised of two groups of patients.

GROUP A - Comprised of children aged 1 to 5 years-given epidural anaesthesia With 21 G hypodermic needle

GROUP B - Comprised of children aged 6-12 years-given epidural anaesthesia with 21 G spinal needle.

METHOD:-

Each patient was examined thoroughly before premedication & induction of anaesthesia. Pulse rate, blood pressure respiratory rate and oxygen saturation were seen and recorded.

PREMEDICATION:-

Group A patients were premedicated with-

- Inj. Glycopyrrolate I/V 0.1 mg to 0.2 mg &
- Inj. Ketamine I/V in the dose of 1 mg/kg

Group B patients were premedicated with

- Inj. Glycopyrrolate I/V 0.1 mg to 0.2 mg &
- Inj. Diazepam I/V 2.5 mg to 5.0 mg or
- Inj. Thiopentone sodium I/V 2-3 mg/kg body weight After premedicating the child, the child was preloaded with isolyte P/Ringer lactate (5-7 ml/kg body weight)

TECHNIQUE

The child was placed in lateral position with knee & hip flexed. Now the back of the child was prepared for epidural injection, using savlon, betadine and spirit. Taking all aseptic precautions, the intervertebral space was identified - the vertebrae corresponding with the highest point of iliac crest being L_4 or the space corresponding with highest point of iliac crest being L_{4-5} . Now after identification of L_{4-5} or L_{3-4} intervertebral space, the 21 G spinal or hypodermic needle, depending upon the age of the patient, was inserted in the desired intervertebral space and advanced. Just when the tip of the needle pierced the subcutaneous tissue the advancement was stopped and a frictionless & leakage free glass syringe filled with 3-5 ml of air was attached to the needle. The needle was advanced into the epidural space by maintaining a pulsatile pressure with the thumb, to detect the loss of resistance. As the tip of the needle entered the epidural space piercing ligamentum flavum, there was a sudden loss of resistance and the piston moved freely forwards in the syringe.

Local anaesthetic solution - 2% lignocaine hydrochloride with adrenaline in the dose of 7-10 mg/kg diluted to 1.5% was injected in the identified epidural space after negative aspiration test for cerebrospinal fluid or blood. Now the patient was immediately made supine and the surgery was allowed to proceed after the establishment of block.

MONITORING :-

After premedicating the child & before performing the epidural block,

the following parameters were noted-

- Pulse
- Blood pressure
- Respiratory rate &
- Oxygen saturation.

Now after performing the epidural block, the above parameters were again noted along with -

- The time taken for onset of block &
- The level of sensory block (Tested by pinching the skin)

Intraoperatively also, the above vitals were noted initially every 5 minutes for upto 20 minutes & then after every 10 minutes till the completion of surgery.

In the postoperative period-

- The duration of postoperative analgesia i.e. duration of sensory block was noted.
- any complication was also noted.



TROLLEY FOR EPIDURAL BLOCK



PART PREPARED AND DRAPED



INSERTING NEEDLE IN
L4-5 INTERVERTEBRAL
SPACE.



IDENTIFYING EPIDURAL
SPACE.



CHILD IN AGONY IN POST OP. PERIOD —
SURGERY PERFORMED IN G.A.



COMFORTABLY SLEEPING CHILD IN POST OP.
PERIOD — SURGERY PERFORMED IN EPI. BLOCK

OBSERVATIONS

OBSERVATIONS

The present work "To see the efficacy, safety and cardiorespiratory stability under epidural anaesthesia in children undergoing various lower abdominal & lower limb surgical interventions" has been made on a series of 60 cases admitted in M.L.B. Medical College Hospital, Jhansi. The following observations have been made.

TABLE NO. -1

AGE & SEX INCIDENCE-

AGE IN YEAR	NO. OF MALE	NO. OF FEMALE	TOTAL NUMBER	%OF MALES	% OF FEMALE
1-5	14	04	18	77.77	22.23
6-12	32	10	42	76.19	23.81

$P < .05$

The maximum number of cases studied, were in the age group of 6-12 years with the male female ratio of 3.3:1 Males predominated, being about 77%. There was significant difference in number of cases in both groups ($p < .05$)

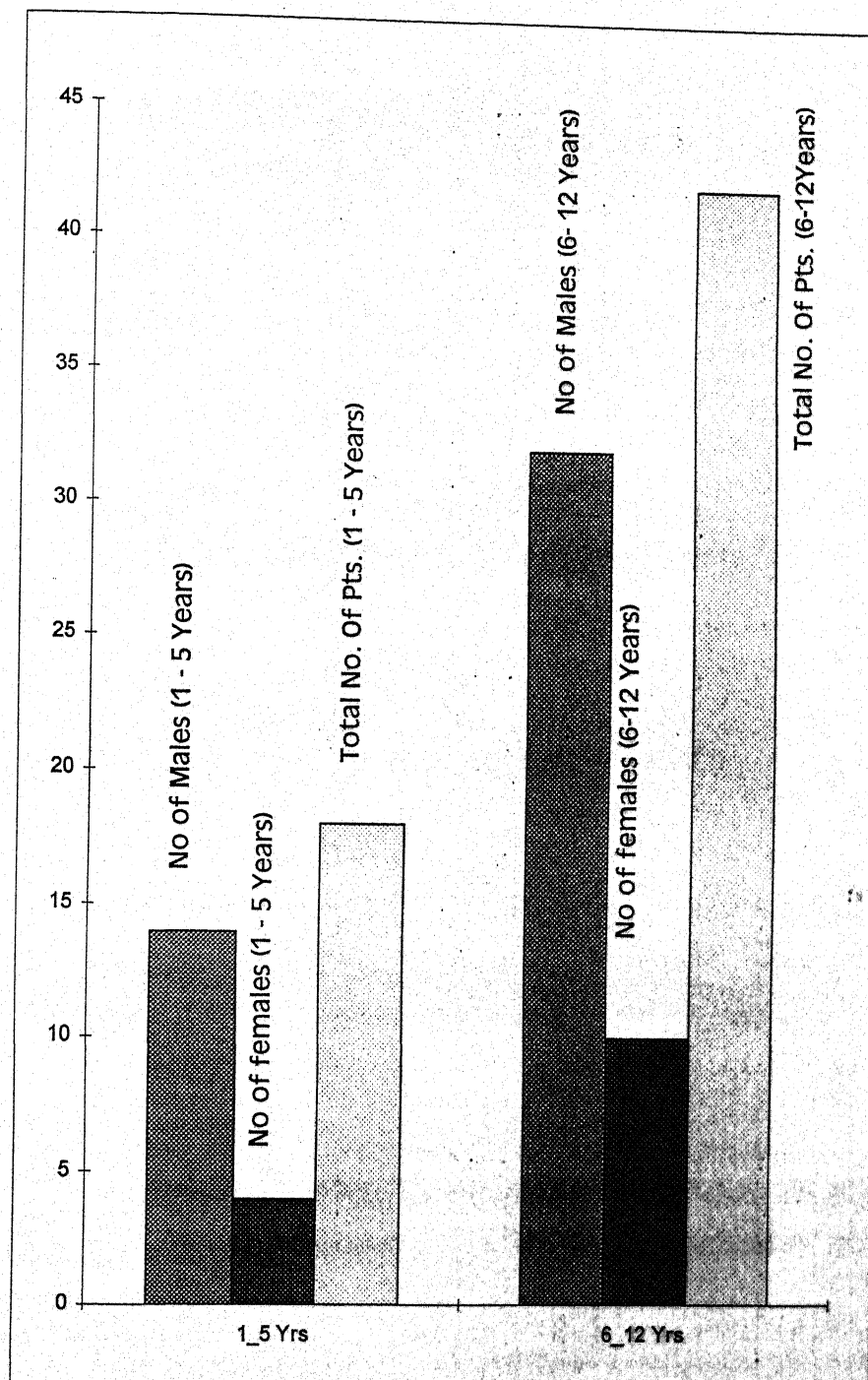
TABLE NO. 2

Mean Weight of patients and mean dose requirement of

Lignocaine hydrochloride with adrenaline on the basis of Weight in both groups:-

GROUP	Mean Weight (Kg) ± SD.	Mean dose (Mg/Kg) ± S.D. (of 2% sol.)	Mean dose (ML) ± S.D. (Of 1.5% Solution)
A	10.88 ± 3.51	8.0 ± 0.66	6.58 ± 2.13
B	20.28 ± 7.26	9.0 ± 0.49	13.50 ± 4.85

Mean Weight in group A was 10.88 Kg with range of approximately 7 to 14 Kg and in group B the mean weight was 20.28 Kg with the range of approximately 13 to 27 Kg. The mean dose on the basis of weight, was 8.0 ± 0.66 mg/Kg in group A and 9.0 ± 0.49 mg/kg in group B. The mean volume of local anaesthetic solution (1.5%) came out to be 6.58 ± 2.13 ml for group A and for group B it was 13.50 ± 4.85 ml.



AGE & SEX INCIDENCE

TABLE NO. - 3

DISTRIBUTION OF OPERATIVE PROCEDURES IN EACH GROUP

OPERATION	NO. OF CASES IN GP. A	NO.OF CASES IN GP. B	TOTAL
-CYSTOLITHOTOMY	2	8	10
-PYEOLITHOTOMY	0	7	7
-URETHROPLASTY	0	3	3
-ORCHIOPEXY	2	1	3
-CORRECTION OF HYPOSPADIAS	3	3	6
-APPENDICECTOMY	1	3	4
-HERNIOTOMY	3	8	11
-K-NAILING FEMUR	0	2	2
-CTEV CORRECTION	5	2	7
-SEQUESTRECTOMY OF TIBIA	0	2	2
-SKIN GRAFTING OF LOWER LIMB.	2	3	5
NET TOTAL	18	42	60

In lower abdominal surgeries the maximum number of cases were of Herniotomy.

In orthopaedic surgeries , the maximum number of cases were of CTEV correction.

TABLE NO.- 4

Mean duration of surgeries in both the groups

Group	Mean \pm S.D. Duration of surgery (in minutes)
A	50.0 \pm 12.82
B	57.77 \pm 30.41

The mean duration of operation was 50.0 \pm 12.82 minutes in group A (1-5 yrs of age) and 57.77 \pm 30.41 minutes in group B (6-12 yrs of age). The duration of operation was more in group B as some long procedures eg. pyelolithotomy , K-nailing femur etc. were exclusively performed in group-B patients.

Table No-5 Shows the level of cooperation in both groups. The level of cooperation was graded as -

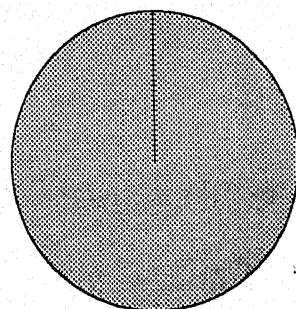
Grade 1- Pts. Showing full cooperation .

Grade 2- Pts showing only some cooperation, requiring moderate sedation or light general anaesthesia prior to the block .

Grade 3- Pts. showing no cooperation at all; all cases requiring light general anaesthesia prior to the procedure.

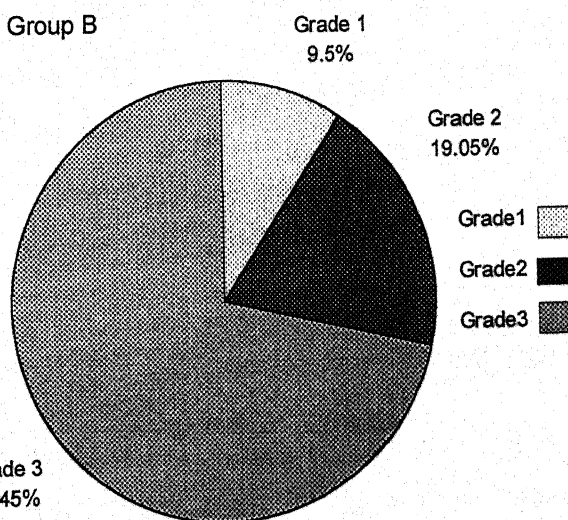
Cooperation was a major problem in most of the patients . 100% of Group A patients belonged to Grade 3 cooperation , all of them requiring light general anaesthesia prior to the procedure while 71.43 %of Gp . B patients belonged to Grade 3 cooperation requiring light general anaesthesia prior to the procedure. The remaining 28.55% of gp . B patients belonged to either Grade 1 or Grade 2 cooperation requiring sedation or light general anaesthesia prior to the procedure .

Group A



Grade 3
100%

Group B



Grade 2
19.05%

Grade 1

Grade 2

Grade 3

Grade 3
71.45%

Distribution Of Patients According to the level Of Cooperation

TABLE NO.5**GRADE OF COOPERATION**

Grade of Coop.	No. of cases in group A	Percentage	No . of cases in group B	percentage
1	0	0	4	9.5
2	0	0	8	19.05
3	18	100	30	71.43

TABLE NO.-6**ONSET OF ANALGESIA IN EACH GROUP :-**

TIME OF ONSET OF SENSORY BLOCK	NO. OF PTS IN GROUP A	MEAN ONSET TIME \pm S.E. IN GROUP A	NO. OF PTS. IN GROUP B	MEAN ONSET TIME \pm S.E. IN GROUP B
0-5 min.	3		6	
5-10 min.	11	9.72 \pm .87	27	9.98 \pm .67
10- 15 Min.	4		6	
15-20	0		1	

$p > .05$

- In the Group A, majority of patients had onset of analgesia within 5 to 10 minutes . The mean was 9.72 \pm 0.87 minutes .
- In the Group B also , majority of patients had onset of analgesia within 5 to 10 minutes . The mean was 9.98 \pm 0.67 minutes .

The maximum time which had been taken for the onset of analgesia was 20 minutes in one case belonging to group B.

Table 7 shows the uppermost level of sensory block achieved in the patients of both groups. The uppermost level achieved in most of the cases was T₉₋₈.

TABLE-7

Uppermost level of sensory block achieved in both groups.

UPPER MOST LEVEL OF SENSORY BLOCK	NO. OF PTS. IN GP. A	NO.OF PTS. IN GP. B
T ₁₀	1	3
T ₉	5	15
T ₈	12	24

TABLE - 8

Changes in pulse rate

GROUP	BEFORE PREMED	AFTER PREMED	IMMIDIATELY AFTER THE BLOCK	INTRA OPERATIVE
A	112.33 ± 21.58	118.24 ± 22.62	118.24 ± 22.62	116.28 ± 21.82
B	94.09 ± 9.72	90.88 ± 8.42	90.88 ± 8.42	90.06 ± 8.22

P>.05

Table No. 8 is showing changes in pulse rate after premedication, immidiately after the block and intraoperatively in both the groups.

Slight increase in the pulse rate after premedication was observed in group A. There was no change in pulse rate immediately after the block and intraoperatively also.

In group B patients,a slight fall or no change at all was observed after premedication.Pulse rate was well maintained immeddiately after the block and intraoperatively also.

There was no clinically significant change in pulse rate.(P>.05)

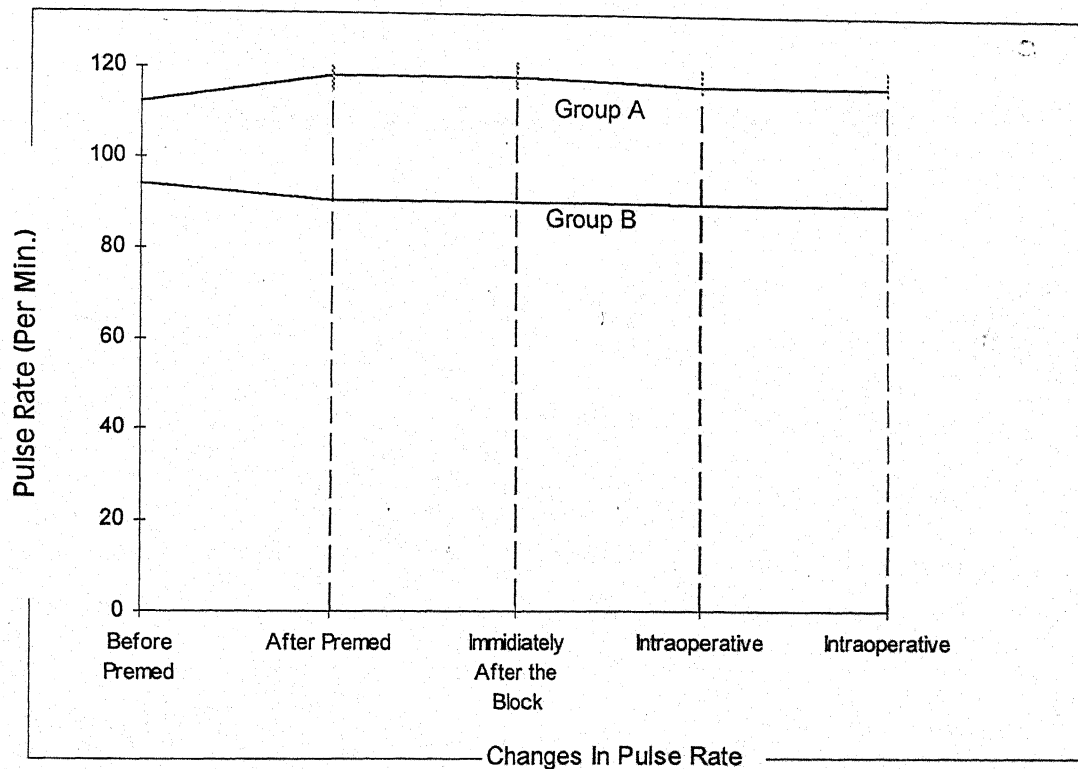


TABLE NO.-9 and 10

Table showing changes in systolic and diastolic blood pressure (mm Hg)

Changes in systolic blood pressure (Mean \pm S.D.)

GROUP	BEFORE PREMED	AFTER PREMED	IMMEDIATELY AFTER BLOCK	INTRAO- PERATIVELY
A	119.44 \pm 16.04	122.21 \pm 15.41	122.21 \pm 15.41	119.21 \pm 15.99
B	114.76 \pm 9.8	110.26 \pm 8.99	110.26 \pm 8.99	109.19 \pm 8.89

P= \geq .05

Changes In diastolic blood pressure (Mean \pm S.D.)

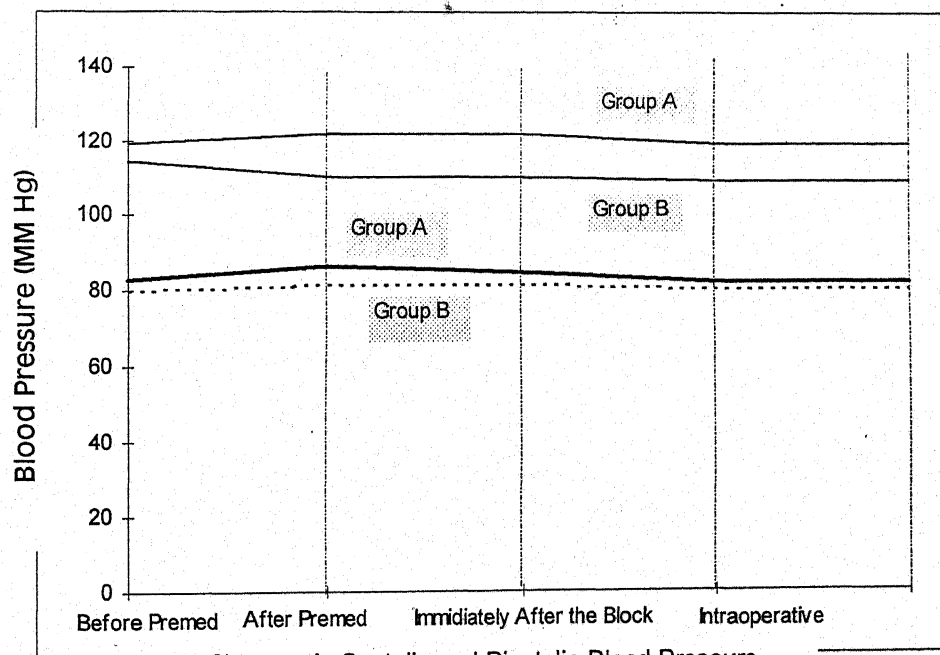
GROUP	BEFORE PREMED	AFTER PREMED	IMMEDIATELY AFTER BLOCK	INTRAO- PERATIVELY
A	80.20 \pm 2.24	81.41 \pm 1.91	81.41 \pm 1.91	80.81 \pm 1.82
B	82.91 \pm 1.86	81.96 \pm 1.90	81.96 \pm 1.90	82.11 \pm 1.72

P $>$.05

Table No.-9 and 10 show change in systolic and diastolic blood pressure after premedication, immediately after the block and intraoperatively throughout the procedure.

Only slight increase in systolic blood pressure in group A patients was observed. Immediately after the block and intraoperatively blood pressure was well maintained to that of basal values. In Group B only slight fall in blood pressure was seen after premedication. Immediately after the block there was no change in blood pressure. Intraoperatively also the blood pressure was well maintained throughout the procedure.

There was no change in diastolic blood pressure after premedication immediately after the block and intraoperatively also. Diastolic blood pressure was well maintained.



Changes In Systolic and Diastolic Blood Pressure

In both groups no significant changes in either systolic or diastolic blood pressure were observed.

The values were not clinically and statistically significant ($p > .05$)

Immediately after the block and intraoperatively also, Diastolic blood pressure was well maintained.

In both groups no significant changes in either systolic or diastolic blood pressure were observed.

The values were not clinically and statistically significant ($p > .05$)

TABLE NO.-11

Changes in respiratory rate (per minute) Mean \pm S.D.

GROUP	BEFORE PREMED.	AFTER PREMED.	IMMEDIATELY AFTER BLOCK	INTRA OPERATIVE
A	16.88 \pm 2.08	16.99 \pm 2.11	16.99 \pm 2.11	16.22 \pm 2.04
B	15.88 \pm 2.44	15.10 \pm 2.12	15.10 \pm 2.12	15.61 \pm 2.11

P $> .05$

Table No.11 shows changes in respiratory rate after premedication , immediately after the block & intraoperatively. In both groups no significant change could be observed as compared to preoperative values .

The values were not clinically as well as statistically significant ($P > .05$)

Table No.- 12 shows changes in oxygen saturation after premedication , immediately after the block and intraoperatively.

Oxygen saturation was well maintained in both groups . No. clinically significant change was observed in oxygen saturation of both groups.

The values were not clinically as well statistically significant .

TABLE - 12Changes in O₂ saturation (%) Mean \pm S.D.

GROUP	BEFORE PREMED	AFTER PREMED	IMMEDIATELY AFTER BLOCK	INTRA OPERATIVE
A	98.0 \pm 1.08	98.0 \pm 1.02	98.0 \pm 1.02	98.0 \pm 1.01
B	98.0 \pm 1.01	98.0 \pm 1.11	98.0 \pm 1.11	98.0 \pm 1.06

P>.05

TABLE NO.-13

Total duration of sensory block

GROUP	MEAN \pm S.D DURATION IN MINUTES	MEAN \pm S.E. DURATION IN MINUTES
A	151.2 \pm 46.8	151.2 \pm 10.84
B	149.8 \pm 37.2	149.8 \pm 5.74

P>.05

Table - 13 shows the total duration of sensory block in both groups.

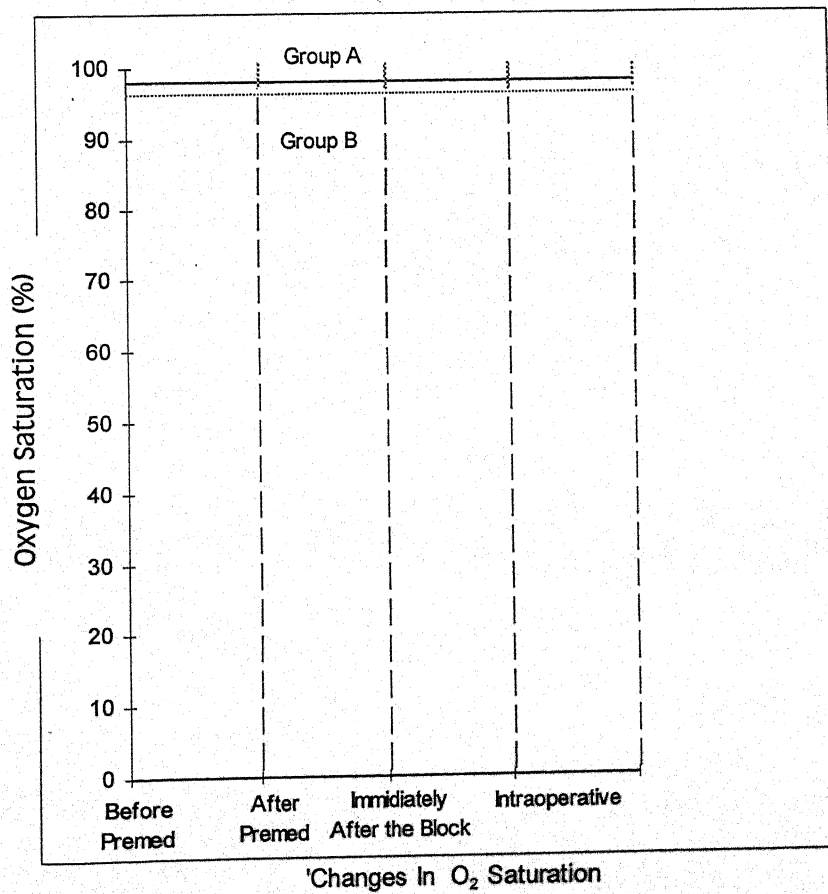
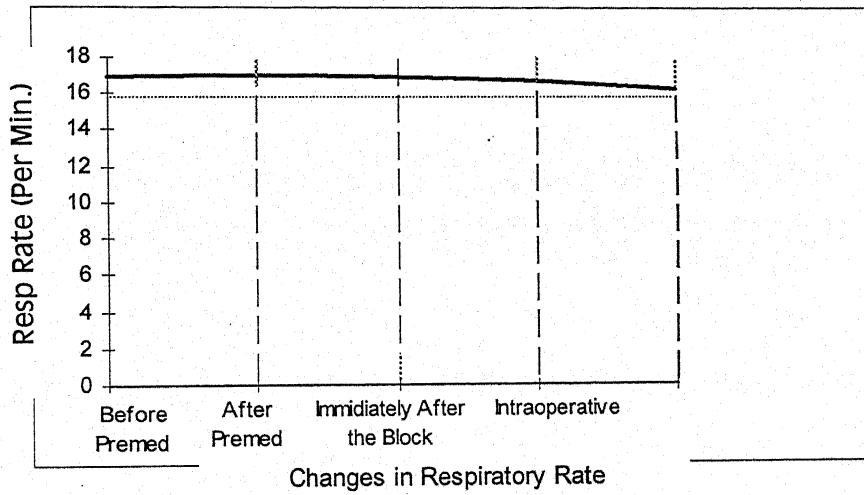
The range of duration of sensory block in both groups was approximately from 100 to 200 minutes with the mean values of 151.2 \pm 46.8 minutes in group A and 149.8 \pm 37.2 minutes in group B.

TABLE NO. - 14

List of complications which were looked for in each group

COMPLICATION	NO. OF CASES IN GROUP A	NO. OF CASES IN GROUP B
DURAL PUNCTURE	-	-
FAILURE OF BLOCK	-	-
ACUTE HYPOTENSION	-	-
NAUSEA & VOMITING	-	2
RETENTION OF URINE	-	-

Not a single complication was observed in Group A . In Group B also no significant complication was noted .Nausea and vomiting in only two cases was seen in Group B which is clinically insignificant.



DISCUSSION

DISCUSSION

Regional anaesthesia was not popular in paediatric patients, but now there has been growing interest for this technique. Epidural anaesthesia can be used for lower abdominal and lower limb surgeries. Regional anaesthesia provides the advantages of reduced requirements for other anaesthetic agents and excellent post operative analgesia. (Yaster M. Maxwell- 1989)

It was only toward the middle of the 1970's that general anaesthesia, used alone, was again seen as having limitations that needed consideration. Timidly at first then almost explosively, the numerous advantages of regional anaesthesia began to be openly reaffirmed again. The resulting redevelopment and acceptance of regional anaesthesia was amplified by the new availability of less traumatic devices and less toxic local anaesthetics, as well as by the considerable scientific advances in anatomy, physiology, and pharmacology as they applied to the use of local anaesthetics- for all age groups and, specifically, for children (Bernard J Dalens 1989).

At a time, when the agent of choice for paediatric anaesthesia was chloroform, the introduction of spinal anaesthesia (Bainbridge, 1900; Gray 1909 a, b, 1910) produced a considerable reduction in morbidity and mortality. Other advantages of note were limitation of anaesthesia to the part to be operated on, muscular relaxation and avoidance of the over distended gut but, more significantly, during the postoperative period there was an almost total absence of vomiting, with an associated rapid return to normal feeding. Gray was also impressed by the long duration of post operative analgesia and the commensurate reduction in the use of opioids.

The introduction of neuromuscular blocking agents to paediatric anaesthetic practice (Rees, 1950) followed by halothane, coincided with a growing controversy over the use of techniques such as spinal an-

aesthesia in children. Some authors continued to extol the technique-"spinal anaesthesia is an excellent method for children" (Berkowitz and Greene, 1951), while others contended that "Spinal anaesthesia in children has been and still is frowned upon by the majority of anaesthetists and surgeons"(Slater and Stephen, 1950)

Following the Woolley and Roe case, it was proposed that all forms of local anaesthesia for major surgery should give place to general anaesthesia (Armstrong Davison, 1965).

Resurgence of interest among paediatric anaesthetist was initially driven by the desire to administer local anaesthetics and opioids in epidural sac for post operative analgesia(Gunter J B, Watcha MF Forestner JE et al 1991).

Later on some employ this approach as the primary anaesthetic for high risk neonates and preterm infants. It was found useful when general anaesthesia is technically difficult or is associated with an increased morbidity and mortality. Regional anaesthesia may offer an alternative to general anaesthesia in children with neuromuscular or metabolic disorder, chronic lung disease, with a history of malignant hyperthermia, and in emergency situations when patients are at increased risk of pulmonary aspiration of stomach contents.(Lynch C Johns RA, Rosen KR, Berkowitz A, LeNeel J.C. , Coiliver J.A. 1985)

In paediatric patients, most regional blocks are performed with the primary goal of providing postoperative analgesia (Rice and Britton, 1993).

The present study has been undertaken encouraged by above studies to see the efficacy, safety and cardiorespiratory stability under epidural block in children undergoing various lower abdominal and lower limb surgical interventions.

The study consisted of 60 patients of either sex, aged 1 to 12 years undergoing lower limb & lower abdominal surgery under lumbar

epidural block.

COOPERATION OF PATIENTS:-

The only problem which was faced in the study was of patient's co-operation. Cooperation of patient was graded as-

Grade 1- Patients showing full cooperation.

Grade 2- Patients showing only some cooperation requiring moderate sedation or light general anaesthesia prior to the block.

Grade 3- Totally uncooperative patients; all cases requiring light general anaesthesia prior to the procedure.

All the patients of group A belonged to Grade 3 co-operation and most of the patients of group B also belonged to grade 3 cooperation. This lack of co-operation had been taken care of by giving light general anaesthesia prior to the procedure.

Children are often frightened by the operating room and personnel wearing gowns & Masks, so they do not cooperate. Light general anaesthesia provides excellent conditions for performing a nerve block and an intervention under regional anaesthesia (Armitage EN. Mc Gown RG 1985).

While lack of cooperation by paediatric patients will never be eliminated, improved sedation agents and the recognition that regional anaesthesia with a light general anaesthetic as sedation is both safe and efficacious has allowed more children to receive the benefit of this approach to balanced anaesthesia (Brown and Schulte-Steinberg, 1988 ; Rice & Britton, 1994 ; Sethna and Berde, 1994)

DOSE OF LOCAL ANAESTHETIC AGENT:-

The mean dose of 2% lignocaine hydrochloride with adrenaline on the basis of weight was 8.0 ± 0.66 mg/kg in group A and 9.0 ± 0.49 mg/kg in group B. The mean volume of 1.5% solution in group A was 6.58 ± 2.13 ml and in group B. it was 13.50 ± 4.85 ml.

With these doses, the uppermost level of sensory block in both the groups was T₉₋₈ in most of the patients, when epidural block was given in L₃₋₄ or L₄₋₅ interspace.

The maximum safe dose for lignocaine hydrochloride with adrenaline is 10 mg/kg for epidural anaesthesia (Isabelle Murat, 1995).

ONSET OF ANALGESIA:-

In present study, the range of onset of analgesia after epidural block was 2 to 15 minutes with the maximum number of patients falling in the range of 5 to 10 minutes. In the patients aged 1 to 5 years, the mean onset time of analgesia was 9.72 ± 0.87 minutes (Mean \pm S.E.) and in patients aged 6 to 12 years of age it was 9.98 ± 0.67 minutes (Mean \pm S.E.) ($P > .05$) Our finding is consistent with-

Onset of analgesia is usually within 5 to 10 minutes with lignocaine hydrochloride with adrenaline (Bernard Dalens 1989)

DURATION OF ANALGESIA:-

Assessment of duration of analgesia in present study after single shot lumbar epidural block with lignocaine hydrochloride with adrenaline shows that the mean duration of analgesia in group A patients was 151.2 ± 10.84 minutes (Mean \pm S.E.) and in group B it was 149.8 ± 5.74 minutes (Mean \pm S.E.).

The approximate range of analgesia in both groups was from 100 to 200 minutes. Our finding is consistent with 'The duration of epidural block with lignocaine hydrochloride with adrenaline is usually 2 to 3 hours' (J.B. Lofstrom and M. Bengtsson 1984)

PULSE RATE AND BLOOD PRESSURE:-

There was no significant effect of epidural block on cardiovascular system. Whatever changes were observed, they were observed after premedication due to the effect of premedicant drugs. In group A, there was a slight increase in the pulse rate and blood pressure due to

the effect of Ketamine while in group B there was slight decrease in the pulse rate and blood pressure (Systolic) due to the effect of diazepam - anxiolytic effect.

The group B patients belonging to Grade 3 cooperation had to be supplemented with sleep dose of thiopentone sodium (2-3 mg/kg) intravenously slowly. There was no effect of thiopentone sodium on cardiovascular system in sleep doses.

After epidural block, pulse rate and blood pressure were more or less constantly maintained - the mean pulse rate preoperatively in group A Patients was 112.33 ± 21.58 per minute and intra-operatively it was 116.28 ± 21.82 per minute. Similarly, in group B, the preoperative mean pulse rate was 94.09 ± 9.72 per minute and intraoperatively it was 90.06 ± 8.22 per minute. There was no significant change in both group ($P > .05$). None of the children, in present study in either group A or group B developed bradycardia intra-operatively or postoperatively as was observed by Welborn LG, Rice LJ 1990 who reported, sedation with Ketamine during spinal anaesthesia resulting in prolonged apnoea with bradycardia in eight of nine infants. The possible explanation for the this phenomna is →Parasympathetic control of the cardiovascular system is well developed at birth, sympathetic control is immature. Decreased sympathetic neural output may explain the normally low blood pressure and their increased susceptibility to reflex bradycardia and hypotension. Furthermore. a low level of baroreceptor activity in infants may reduce their ability to adapt to hypotension by an increase in heart rate. Absence of response in our patients may be due to premedication with glycopyrrolate or because of age factor.

The mean systolic blood pressure in group A in the preoperative period was 119.44 ± 16.04 mm Hg and intraoperatively it was 119.21 ± 15.99 mm Hg, Similarly in group B, the mean systolic blood pressure preoperatively was 114.76 ± 9.8 mm Hg and intraopertively, it was 109.19 ± 8.89 mm Hg. Thus no significant change in systolic blood pressure

was observed in both groups ($p > .05$).

The mean diastolic blood pressure in group A in the preoperative period was 80.20 ± 2.24 mm Hg and intra operatively it was 80.81 ± 1.82 mm Hg. Similarly in 2group B, the mean diastolic pressure in preoperative period was 82.91 ± 1.86 mm Hg amd intraoperatively it was 82.11 ± 1.72 mm Hg. There was no change in diastolic pressure in both the groups.

Excellent haemodynamic stability may be attributed to-

1. Lower resting sympathetic tone (Sethnan N.F., Berde C.B. 1994)
2. Greater ability to compensate for a fall in systemic vascular resistance.(Sethnan N.F., Berde C.B. 1994)

Our findings are consistent with-

Haemodynamic alterations are not usually seen in children (Murati, Delleur MM. Esteve C, Egu JF, Raynaud P, Saint Maurice C 1987)

Epidural anaesthesia in children provides for haemodynamic stability during surgery. (Salerno & others 1989; Atallah and others 1993; Nakamura & Takasaki 1991)

RESPIRATORY RATE AND OXYGEN SATURATION:-

In the present study, none of the cases showed any significant change in respiratory rate and oxygen saturation in any of the group.

The mean respiratory rate in group A preoperatively was 16.88 ± 2.08 per minute, while after epidural block intraoperatively it was 16.22 ± 2.04 per minute Similarly in group B, the preoperative mean respiratory rate was 15.88 ± 2.44 per minute (Mean \pm S.D.) while after epidural block intraoperatively it was 15.61 ± 2.11 per minute. Thus no significant change in respiratory rate was observed in both groups ($p > .05$)

There is no evidence of intraoperative or postoperative apnoea in present series of patients as was observed by Welborn LG; Rice LJ;

Broadman LM; Ruttimann UE; FinK R. 1990, who reported

Postoperative apnoea with bradycardia in can follow caudal anaesthesia without opioids in the preterm infants.

The patients of present series were of 1 to 12 years of age group.

The oxygen saturation in both the groups was well maintained to 97% to 98% in all the cases in group A the mean oxygen saturation preoperatively was 98.0 ± 1.08 percent while after epidural block intraoperatively it was 98.0 ± 1.01 percent. Similarly in group B, the mean oxygen saturation preoperatively was $98.0 \pm 1.01\%$ while after epidural block intraoperatively it was 98.0 ± 1.06 percent. Thus no significant change in oxygen saturation was observed in any of the groups ($p > .05$).

In a normal child, respiratory depression does not occur unless the block extends up to the cervical dermatomes and paralyzes the diaphragm. (Abajian JC, Mellish RWP, Browne AF, Perkins FM, Lambert DH, Mazuzan JE 1984)

COMPLICATIONS:

We observed the following complications in our study.

1. DURAL PUNCTURE:

No episode of dural puncture was observed in both groups as the blocks were very carefully performed using frictionless & leakfree glass syringes for detection of loss of resistance.

Dural puncture may be seen when inappropriate equipment is used, if the loss of resistance technique is not used correctly or when there is poor control of needle penetration into tissues (Bernard Dalens 1989).

2. FAILURE OF BLOCK:

The block was successful in all the patients of both groups.

3- ACUTE HYPOTENSION:

Acute hypotension was not seen even in a single patient of either

group. Infact even gradual hypotension (ie. a change of 30 percent from the preoperative blood pressure) was also not observed in both groups.

Hypotension is not usually seen in children (Murat I, Delleur MM; Esteve C; Egu JF, Raynaud P; Saint Maurice C 1987), even if associated with an inadvertent total Spinal injection (Dalens 1989)

4. NAUSEA AND VOMITING:

Nausea and vomiting was not observed in any case belonging to group A.

In group B, nausea and vomiting was observed in 4.76% of patients which is clinically insignificant. These were of herniotomy- probably the release of serotonin due to bowel handling was the cause of nausea & vomiting.

Using epidural block, there is an almost total absence of vomiting during the post operative period (D.S. Arthur & L.R. Mc Nicol 1986)

5. RETENTION OF URINE:

Retention of urine was not observed in any case of both groups.

Sphincter disturbances after epidural block are usually not observed in children (Dalens 1989)

Thus in this study, we have seen that epidural block in children for Lower abdominal and Lower limb surgeries can be performed without any hazards. Requirement of general anaesthetic agents is minimised thus avoiding harmful effects of general anaesthesia.

CONCLUSIONS

CONCLUSIONS

On the basis of observations made on 60 cases, studied in the present Series, following conclusions are drawn-

1. Procedure is economical for the patient.
2. Epidural anaesthesia in children is quite safe and effective method for lower abdominal and lower limb surgery.
3. Cardio - respiratory Stability is excellent with this technique.
4. Patient wakes painfree after surgery is over - child is much easier to manage and at the same time the anxiety of attendants becomes much less as the patient lies comfortably post operatively.
5. Many of the harmful effects of general anaesthesia when general anaesthesia is used as a sole anaesthetic technique, can be avoided by using epidural block.
6. Side effects or complications of the technique itself are negligible.
7. As most of the patients in this age group are uncooperative, they can be supplemented with light general anaesthesia prior to the procedure.
8. Acceptance of technique is not affected by Sex.
9. The mean dose of 1.5% lignocaine hydrochloride with adrenaline in children aged 1 to 5 years is 4.45 to 8.71 ml while in 6 to 12 years age group, it is 8.65 to 18.35 mL. With these doses, the upper most level of sensory block in most of the Patients is T_{9-8} , when the block is given in L_{3-4} or L_{4-5} interspace.

BIBLIOGRAPHY

BIBLIOGRAPHY

1. Armstrong Davison, M.H. The evolution of Anaesthesia, (1965); p. 176.
2. Bailey PW, Median epidural septum and multiple cannulation. Anaesthesia (1986); 41: 881-882.
3. Bainbridge W.B. Analgesia to children by spinal injection with a report of new method of sterilization of the injection fluid. Medical Record (1900); 58:937
4. Batnitzky S, Keucher TR, Mealey Jr J, Campbell RL. Iatrogenic intraspinal epidermoid tumors J Am, Med. Assoc. (1977); 237: 148-150.
5. Berkowitz S, and Greene, B.A. Spinal anaesthesia in childdren, report based on 350 patients under 13 years of age. Anaesthesiology (1951); 12:376.
6. Blomberg RG, Olsson S. The lumbar epidural space in patients examined with epidurocopy, Anesth. Analg. (1989);. 68: 157-160.
7. Bokesch PM, Castaneda AR, Ziemer G, Wilson JM. The influence of a right to left shunt on lidocaine pharmacokinetics. Anaesthesiology (1987);. 67:739-744.
8. Bosenberg AT, Bland BAR, Schulte Steinberg O, Downing JW. Thoracic epidural anaesthesia Via caudal route in infants. Anaesthesiology (1988);. 69:265-269.
9. Bricker SRW, Telford RJ, Booker PD. Pharmacokinetics of bupivacaine following intraoperative intercostal nerve block in neonates and in infants aged less than 6 months. Anaesthesiology (1989); 70 : 942 - 947.
10. BROMAGE PR. Unblocked segments in epidural analgesia for relief of pain in labour. Br. J.Anesth (1972);. 44:676-679.
11. Bromage PR. Anatomy In : Bromage PR, ed Epidural Analgesia

Philadelphia : Saunders, (1978); : 8-67. (Quoted from 'Regional Anaesthesia in infants, children & Adolescents' by Bernard Dalens)

12. Bromage PR. Identification of epidural space. Philadelphia : Saunders; (1978), 176-214. (Quoted from 'Regional Anaesthesia in infants, children & Adolescents' by B.Dalens)
13. Cheng PA. The anatomical and clinical aspects of epidural anaesthesia. *Anesth Analg* (1963);. 42:398-415.
14. Colliver JA, Marrero P.C., Roush JR, Manchikanti L : Assessment of age related acid aspiration risk factors in pediatric, adult, and geriatric patients. *Anesth*, (1985); 64:11-17.
15. Cousins MJ. Haematoma following epidural block. *Anaesthesiology* (1972); 37:263-264.
16. Cousins MJ, Bromage PR. Epidural neural blockade. Neural blockade in clinical anaesthesia and management of pain. Second ed philadelphia : Lippincott; (1988); 253-360 (Quoted from ' Regional Anaesthesia in infants, childrens & adolescents' by B. Dalens)
17. Dalens B, Bazin JE, Haberer J.P. Epidural bubbles as a cause of incomplete analgesia during epidural anaesthesia. *Anaesth Analg* (1987); 66: 679-683.
18. Dalens B, Haberer J.P. Epidural anaesthesia in children. *Anaesthesiology* (1987) 66:614-615.
19. Dalens B, Regional anaesthesia in children. *Anesth Analg* (1989) 68:654.
20. Dalens B, Hasnouai A : Caudal anaesthesia in pediatrics surgery. *Anesth Analg* (1989); 68:83.
21. Dalens B, Intervertebral epidural anaesthesia in paediatric surgery. *Pediatr Anaesth* (1991); 1:107-117.
22. Defalque RJ. Exaggerated spread of epidural block. *Anaesthesiology* (1967); 28:229-231.

23. Denson DD, Coyle DE, Thompson GA, Myers JA. Alpha -1-acid glycoprotein and albumin in human serum bupivacaine binding. Clin Pharmacol Ther (1984); 35 : 409-415.
24. Difazio CA, Metabolism of local anaesthetics in the fetus, newborn and adult, Br J.Anaesth (1979); 51:29-36.
25. D.S. Arthur and L.R. Mc Nicol Br. J.Anaesth. (1986); 58, 760-778.
26. Eather KF : Regional Anaesthesia for infants and children Int. Anesthesiol Clin. (1975) 13:19.
27. Elander G and others : pain relief in infants after Major surgery : A descriptive study, J. Pediatr Surg (1991); 26:128.
28. Eyres RL, Bishop W, Oppenheim RC. Brown TCK. Plasma lignocaine concentrations following topical laryngeal application. Anaesth Int. Care (1983); 11:23-26.
29. Eyres RL, Bishop W, Oppenheim RC, Brown TCK, Plasma bupivacaine Concentration in children during caudal epidural analgesia. Anaesth Intens Care (1983); 11:20-22.
30. Eyres RL, Hastings C, Brown TCK, Oppenheim RC. Plasma bupivacaine concentrations following lumbar epidural anaesthesia in children. Anaesth Intens Care (1986); 14:131-134.
31. Farr RE : Local anaesthesia in infancy and childhood. Arch Pediatr (1920); 37:381.
32. Goresky GV : Post operative pain management for children. Anaesthesiol Clin North Am (1991); 9:801.
33. Gray H.T. (1909 a) A study of spinal anaesthesia in children and infants. Lancet, 913.
(1909 b) A study of spinal anaesthesia in children and infants. Lancet, 991.
(1910) A further study of spinal anaesthesia in children Lancet,

1611.Quoted from Br. J. Anaesth (1986)

34. Gunter J.B., Watcha MF, Forestner JE, et al. Caudal epidural anaesthesia in conscious premature and high risk infants. Journal of pediatric surgery (1991); 26:9-14.
35. Hannallah RS, Epstein BS : The Pediatric patient, Anaesthesia for ambulatory surgery, PHILADELPHIA (1991). Quoted from Smith's Anaesthesia for infants & children.
36. Heldt TJ. Negative pressure in epidural space. Am J Med Sci (1928); 175 : 371-375.
37. Hilt H, Gramm HI, Link J. Changes in Intracranial pressure associated with extradural anaesthesia, Br. J Anaesth (1986); 58:676-680.
38. Hodgkinson R.Total spinal block after epidural injection into an interspace adjacent to an inadvertent dural perforation. Anaesthesiology (1981); 55:593-595.
39. Jansen E. Dtsch Z Nervenheilk (1926); 94: 280-285.
40. Jirout J. Dynamics of the spinal dural sac under normal conditions. Br J.radiol (1967); 40:209-213.
41. Langer JC and others. Intraoperative bupivacaine during outpatient hernia repair in children J.Pediatr Surg. (1987) 22:267.
42. Lee Neel J.C. Meignier M,Souron : Postoperative dorsal epidural analgesia in the child with respiratory disabilities. Anaesthesiology (1983); 59:473-475.
43. Lerman J,Strong HA. Le Dez KM, Swartz J, Rieder MJ, Burrows FA. Effects of age on the serum concentration of alpha - 1- acid glycoprotein and the binding of lidocaine in pediatric patients. Clin Pharmacol Ther (1989); 46:219-225.
44. Lerman J, Burrows F.A. LeDez KM, strong A. Alpha-1acid glycoprotein and the binding of lidocaine in children with congenital heart disease. Can J Anaesth (1990); 37:883-888.

45. Liu P, Feldman HS, Covino BM, Giasi R, Covino BG. Acute cardiovascular toxicity of intravenous amide local anaesthetics in anaesthetized ventilated dogs. *Anesth Analg* (1982); 61:317-322.
 46. Morishima HO, Pedersen H, Finster M, Sakuma K, Bruce SL, Gutsche BB, Stark RI, Covino BG. Toxicity of lidocaine in adult, newborn and fetal sheep. *Anaesthesiology* (1981); 55:57-61.
 47. Munson ES. Etidocaine, bupivacaine and lidocaine seizure thresholds in monkey. *Anaesthesiology* (1975); 42:471-478.
 48. Odom JA, Sih LL. Epidural analgesia and anticoagulant therapy experience with one thousand cases of continuous epidurals. *Anaesthesia* (1983); 38:254-259.
 49. Ramsey M, Roberts C. Epidural injection does cause increase in CSF pressure. *Anesth Analg* (1991); 73:668.
 50. Rees G.J. Anaesthesia in the newborn. *Br. Med. J.* (1950) 2:1419.
 51. Rice LJ, Hannallah RS: Local & regional anaesthesia. *Smith's Anaesthesia for infants and children* ed 5 st. Louis (1990) Quoted from *Smith's Anaesthesia for infants & children*.
 52. Rice LJ, Britton JT: Neural blockade for pediatric pain management. *Acute pain, mechanisms and management*. St Louis, (1992).
 53. Rice LJ, Britton JT: Pediatric post operative analgesia. *Semin Anesth* XII : 27, (1993).
 54. Rice LJ, Britton JT : Pediatric regional anaesthesia. *Advances in anaesthesia* vol. II St. Louis, (1994).
- 51-54 are quoted from *Smith's Anaesthesia for infants & children*.
55. Ruston FG. Epidural anaesthesia in paediatric surgery. *Anesth Analg* (1957); 36:76-79.
 56. Savolaine ER, Pandya JB, Conover SR. Anatomy of the human lumbar epidural space. *Anaesthesiology* (1988); 68:217-220.

57. Schulte-Steinberg, O, In neural blockade in clinical anaesthesia and management of pain, 1st Edn. (1980) P-506.
 58. Schulte Steinberg O, Regional anaesthesia for children. Ann Chir Gynaecol (1984); 73:158-165.
 59. Schulte Steinberg O Brown TCK : Neural blockade for paediatric surgery ed 2, Philadelphia (1988).
- 58 & 59 are quoted from ' Regional Anaesthesia in infants, children & adolescents' by B.Dalens.
60. Scott DB, Cousins MJ : Clinical pharmacology of local anaesthetic agents : Neural blockade, ed 2. Philadelphia, (1988).
- Quoted from Smith's Anaesthesia for infants and children.
61. Scott DB : Regional anaesthesia for pediatric Surgery. Analg (1989); 69:697.
 62. Sechzer PH. Subdural space in spinal anaesthesia Anaesthesiology (1963); 24:869-870.
 63. Sethna and Berde C.B. : Pediatric regional anaesthesia. Pediatric anaesthesia ed 2 New York (1988). Quoted from Smith's anaesthesia for infants & children.
 64. Sethna NF Berde CB: Pediatric regional anaesthesia. Pediatric New York (1994) Quoted from Smith's Anaesthesia for infants and children.
 65. Sethnan NF, Berde C.B. Paediatric Regional Anaesthesia. New York, churchill Livingstone 3rd ed (1994); 281-287.
 66. Slater HM, and Stephen, C.R. Hypobaric pentocaine spinal anaesthesia in children. Anaesthesiology (1950); 11,709.
 67. Usubiaga JE, Maya F Usubiage LE. Effect of thoracic and abdominal pressure changes on the epidural space pressure. B.T. J. Anaesth (1967);. 39:612-618.
 68. Watcha MF, Thach BT, Gunter J.B. Postoperative apnea after caudal

anaesthesia in an ex-premature infant. *Anaesthesiology* (1989); 71:613-615.

69. Welborn LG, Rice LJ, Hannallah R.S., Broadman LM, Ruttimann UE. Fink R. Postoperative apnea in former preterm infants : prospective comparison of spinal and general anaesthesia. *Anaesthesiology* (1990); 838 - 842.
70. Wood M. Plasma drug binding : Implications for anaesthesiologists. *Anesth Analg* (1986); 65:786-804.
71. Yaster M. Maxwell L.G. Pediatric regional anaesthesia *Anaesthesiology* (1989); 70; 324-338.
72. Zarzur E. Anatomic studies of the human lumbar ligamentum flavum. *Anesth Analg* (1984); 63:499-502.